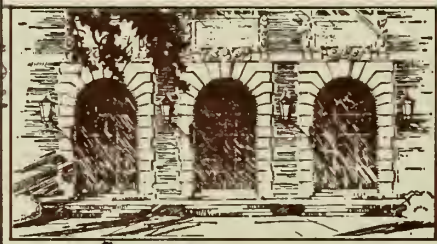
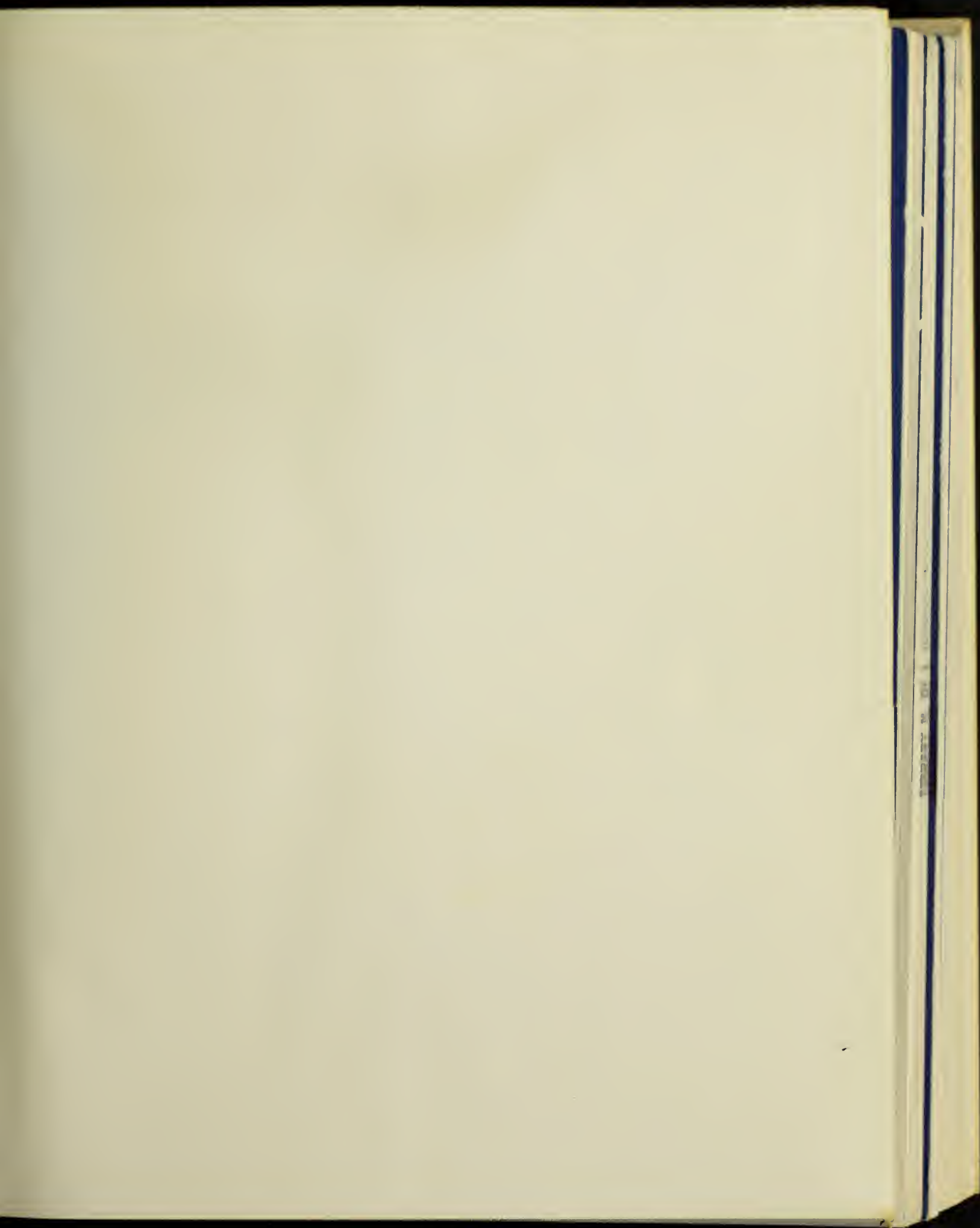
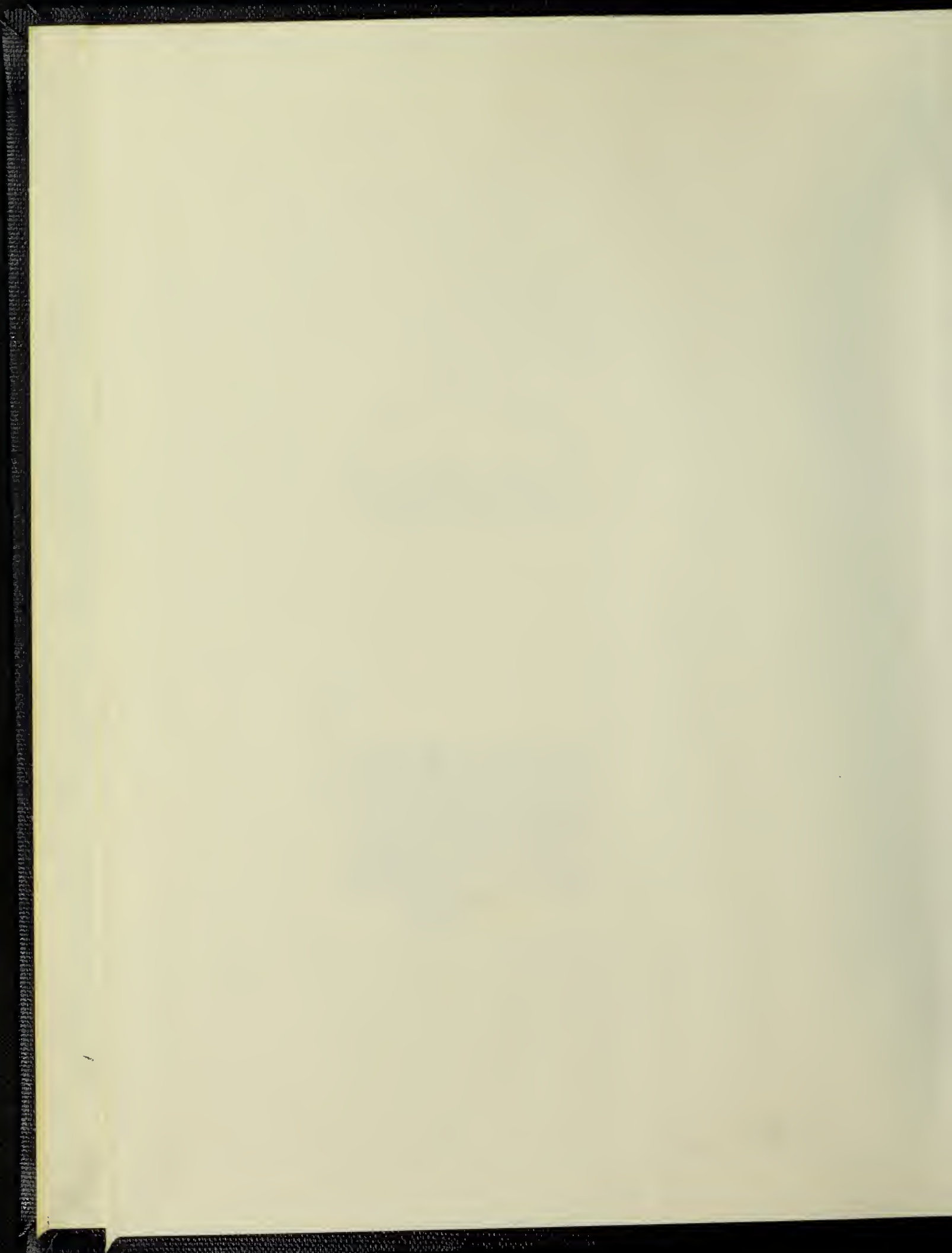


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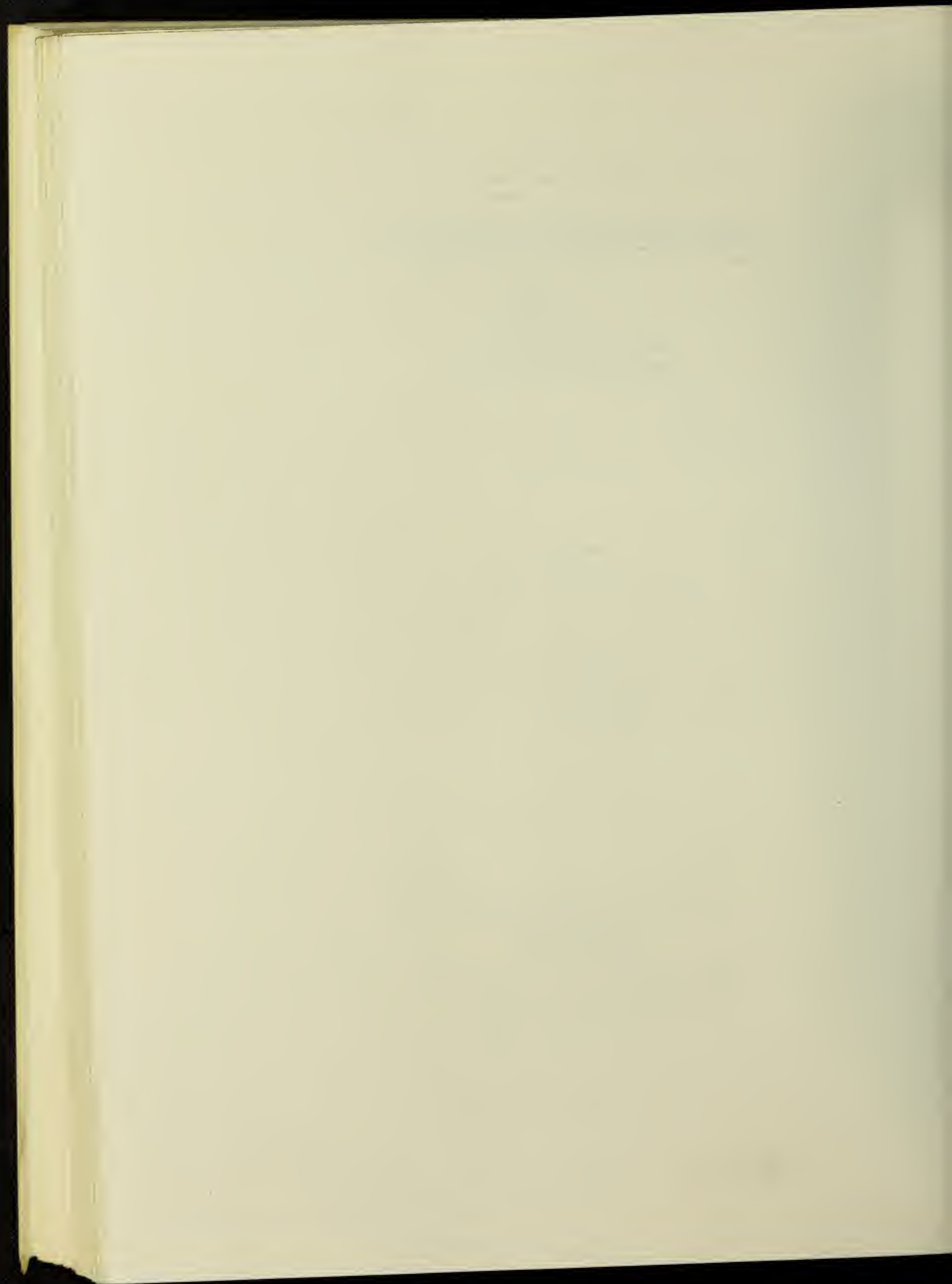
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PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
ln.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotrophic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10 ⁻⁹)
CNS	central nervous system	pg	picogram (10 ⁻¹²)
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
µg	microgram(s)	resp.	respectively
hr	hour(s)	RNA	ribonucleic acid
i.m.	intramuscular	s.c.	subcutaneous
i.p.	intraperitoneal	sec	second(s)
IU	international unit(s)	U	unit(s)
i.v.	intravenous	UV	ultraviolet
kg	kilogram(s)	WBC	white blood cells (leukocytes), white blood count
LD ₅₀	median lethal dose(s)	wk	week(s)
m	meter(s)	wt	weight(s)
M	molar	yr	year(s)
mEq	milliequivalent(s)		
mM	millimolar		
µM	micromolar		
mC, µC	milli-,microcurie(s)		



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3601 PRODROMAL DYSIMMUNITY IN CANCER. PATHOPHYSIOLOGY OF PRE- AND PERINEOPLASTIC IMMUNE DEFICIENCY DISORDERS THAT MAY REPRESENT SYNDROMES OF TRANSITION FROM DYSIMMUNITY TO CANCER. (E.) De Carvalho, S. (Belmont Med. Clin., Bellflower, Calif.). *Oncology* 28(1):1-34, 1973.

Instances of immune disorders which are apparently prodromic to the development of malignancy are described. These include one case of pulmonary granulomatous asbestosis ending in IgA myeloma, two cases of Sézary syndrome with lymphocytic lymphoma, one case of Dühring's disease with lymphoma, and three cases of hepatobiliary diseases developing hepatoma and skin carcinomas. The pathophysiology is discussed in the light of the transition between the dysimmunity and the neoplasia. Postulates evolving from these observations are: (a) the existence of tissue or organ-oriented T-cell surveillors (histiosophocytes), (b) obligatory antecedent dysimmunity in cancer, (c) therapy must be directed to the dysimmune situations rather than empirical augmentation of 'cancer immunity'. All the transition syndromes presented four stages of immunologic evolution: (1) the initial stage of immune competence; (2) a stage of immune dysfunction, asymptomatic although deficiencies can be demonstrated; (3) a stage of overt, clinical, immunologic deficiency (prodromic, preneoplastic) when surveillance is inappropriate; and (4) a stage of clinical manifestations of the malignancy (immunodecadence) with persistence of the immunologic disorder (perineoplastic). Allogenic carcinogenesis and its potentiation by immunosuppression is recognized as a fourth category of carcinogenic agent next to chemicals, radiations, and viruses. (119 references)

3602 ROLE OF THE MEGAKARYOCYTE AND PLATELET IN THE LEUKEMIC PROCESS IN MICE AND MEN - A REVIEW AND HYPOTHESIS. (E.) Brodsky, I. (Hahnemann Med. Coll., Philadelphia, Pa.). *J Natl Cancer Inst* 51(2):329-335, 1973.

Electron microscopy has revealed that the C-type virus particles of murine and feline leukemias are characteristically in the cytoplasm of megakaryocytes and platelets. In mice, thrombocytopenia can be used in dose-response curves to titer both Friend and Rauscher viruses. At 2 wk after infection with Rauscher virus (RV) the bone marrow is hypercellular with megakaryocytic hyperplasia associated with thrombocytopenia but with no infiltration with leukemic cells. Within 4-6 wk in the surviving mice, recovery is spontaneous. However, 10-24 wk after infection thrombocytopenia again develops, but is now associated with lymphocytosis in the peripheral blood and leukemic cell infiltration of the bone marrow. Metabolic studies indicate that the initial thrombocytopenia is due to a direct effect of the virus on platelet metabolism. This preleukemic state, particularly in regard to thrombocytopenia, can also be demonstrated in spontaneous AKR murine leukemia. Fortuitous observations in humans have shown that thrombocytopenia is a major preleukemic manifestation for this species as well. The AKR mouse probably represents the best experimental model for human lymphoproliferative disorders. While human patients recover from an initial thrombocyto-

penia with megakaryocytic hyperplasia, only to again develop thrombocytopenia with onset of acute leukemia, also in the AKR mouse the second phase of thrombocytopenia is associated with onset of leukemia. Since many human viruses can cause severe thrombocytopenia, the possibility is suggested that such thrombocytopenia-inducing viruses might be potentially leukemogenic in individuals with appropriate genetic predisposition for the development of leukemia. Rubella virus is cited as a possible example. It is even conceivable that most preleukemic states are entirely reversible. Infectious mononucleosis, a disease most likely caused by a herpesvirus, might well fit into such a category. With the foregoing considerations in mind, a study was initiated on platelet kinetics in chronic myeloproliferative disorders, namely, polycythemia vera (PV), myelofibrosis and myeloid metaplasia (MF), and chronic myelocytic leukemia (CML). PV is a preneoplastic state which frequently evolves into MF and in 10-15% of the cases terminates in acute leukemia. CML is a full-blown leukemic condition considered a close relative of PV. Platelet and fibrinogen studies utilizing ⁷⁵Se revealed that platelet as well as fibrinogen survivals were significantly longer in CML compared to PV or MF. It was postulated that platelets produced in patients with CML are functionally abnormal and underutilized as compared to patients with PV and MF. The relative overutilization of platelets, when associated with an increased turnover of fibrinogen is probably a major factor in explaining the increased incidence of thrombosis in PV and MF and perhaps the associated hemorrhage. The preceding observations indicate that the megakaryocyte and platelet are important factors in the pathogenesis of both murine and human leukemia. Awareness of the preleukemic state raises the question of whether leukemia is a true neoplasm and whether the current approach to leukemia therapy based on the neoplastic theory is appropriate. (34 references)

3603 GENETIC STUDIES ON RNA TUMOUR VIRUSES AND THEIR HOSTS. (E.) Martin, G. S. (Imperial Cancer Res. Fund Lab., London, England). *Br Med Bull* 29(3):241-246, 1973.

Genetic studies on the avian and murine RNA tumor viruses are reviewed. Functions of the viral genome are first discussed before two models of the structure of the genome are described. One model supposes that each subunit is genetically distinct and, since each gene is represented only once, the viral genome is haploid. On this model, simultaneous or successive deletions in each subunit would be necessary to generate a non-transforming virus. On the second model, the subunits of the viral RNA are genetically identical and, since each gene is represented several times, the genome is polyploid. Some of the genetic factors that control the susceptibility of the host cell to infection by an RNA tumor virus affect the ability of the virus to penetrate into the host cell while others affect subsequent intracellular events in the replication cycle of the virus. In chickens, four loci, each affecting susceptibility to a specific virus subgroup have been identified. In the mouse, loci governing the synthesis of surface receptors have not been found. The frequency

of induction of endogenous virus, both *in vivo* and *in vitro*, is subject to genetic control. Partial expression of the endogenous viral genome can also occur; cells which are not producing complete virus can contain some of the gene products specified by the endogenous virus. It has not yet been reported whether endogenous viruses induced *in vitro* are tumorigenic *in vivo*. However, it is likely that at least some genetically transmitted viruses are oncogenic. (64 references)

- 3604 CHEMICAL CARCINOGENESIS. A NEW APPROACH TO THE MOLECULAR AND CELLULAR MECHANISMS. (E.) Mekler, L. B. (Inst. Exp. Clin. Oncol., USSR Acad. Med. Sci., Moscow). *Oncology* 28(1):63-82, 1973.

Published data on molecular and cellular phenomena observed in chemical carcinogenesis and during the formation and turnover of macromolecules of normal cell plasma membrane at different phases of the cell cycle are collated. The principal peculiarities of chemical carcinogenesis are: (1) hereditary nature of cell transformation; (2) absence of full correlation between mutagenic and carcinogenic activity; (3) the obligatory formation of a covalent bond between the chemical carcinogen and the macromolecule target; (4) the carcinogen's ability to bond with H protein(s); (5) the absence of H protein(s) in transformed cells; (6) the induction of passivity of the overwhelming mass of cells of organs and tissues sensitive to the chemical carcinogen and subject to it, and the reversibility of this passivity in all affected cells, except transformed ones; (7) the different immunological specificity of new transplantation antigens induced in cells of a single clone by one and the same carcinogen. These principal peculiarities can be understood from a single standpoint if it is assumed that the key events during chemical carcinogenesis are the alterations of the conformation of certain proteins of plasma, and perhaps other cell membranes, which are coupled to a loss of reversibility of their conformational transitions. As a result of the peculiarities of the formation and turnover of cell membrane macromolecules, which are characteristic of resting and dividing cells, these alterations of conformation are inherited according to Sonneborn's heredity mechanism only by cells that have emerged from the G_0 phase of the cell cycle. The malignant cell which has arisen during chemical, or some other types of carcinogenesis, is thus locked in the G_1 -phase of the cell cycle, and therefore is continuously dividing, while bypassing the G_0 phase. (116 references)

- 3605 CONJECTURES ON SUPERCONDUCTIVITY AND CANCER. (E.) Marton, J. P. (Ctr. Interdisciplinary Studies Chem. Physics, U. Western Ontario, London, Canada). *Physiol Chem Physics* 5(3):259-270, 1973.

On the assumption that biological cell membranes possess superconductive properties and that dead and cancerous cells do not, four hypotheses are derived, which are used to model the growth of tissues. Effects of the geomagnetic field on the superconductive processes are examined, and the results are compared with cancer

mortality data over a large part of the world. The conclusions do not appear to violate any physical principles and are not in conflict with available experimental data. (18 references)

- 3606 CANCER GENETICS, PART II: STUDIES IN HUMANS. (E.) Lynch, H. T. (Creighton U. Sch. Med., Omaha, Nebraska). *Nebr Med J* 58(11):401-407, 1973.

Studies in human cancer genetics have been hampered significantly by inaccurate reporting of cancer diagnoses. Furthermore, the patient must be viewed in context with myriad genetic and non-genetic factors and their interactions, especially the patient's environmental exposures. Statistics on cancer incidence have shown that wide variations in the frequency of specific anatomic varieties of cancer from one population to the next is the rule. The association of genetic disorders with cancer incidence such as testicular cancer with testicular maldevelopment and testicular feminization syndrome is reviewed, and a listing of cancerous and precancerous diseases with either a clearcut or suggested hereditary etiology is presented. (24 references)

- 3607 A NEW LOOK AT THE CELL SURFACE. (E.) Doljanski, F. (Hebrew U.-Hadassah Med. Sch., Jerusalem, Israel). *Israel J Med Sci* 9(3):251-257, 1973.

Recent developments which contribute to the understanding of the dynamic nature of the cell surface in higher animals are discussed. Cell interactions are characterized by a highly ordered pattern of cell behavior, whereas disordered patterns of behavior, invasiveness and ability to metastasize, are unique to the malignant neoplastic cell. Using proteolytic enzymes, it has been demonstrated that the cell surface contains a heterogeneous population of glycoproteins and mucopolysaccharides, carrying different antigenic determinants such as histocompatibility and blood group antigens and receptor molecules for drugs. Cancer cells show alteration of these surface constituents. Studies on the interaction between cells and plant agglutinins suggest that neoplastic cells possess a certain cell surface microstructure not possessed by normal cells, which allows for increased agglutinability, although the situation is not as clear-cut as was initially suggested. Work with lectins indicating a system of possible control by "outside" surface molecules on intracellular and nuclear functions is described. Other studies on the kinetics, biosynthesis and turnover of cell surface components demonstrate that surface membranes of dividing and non-dividing cells appear to be in a similar, continuous active state of synthesis *in vivo* and *in vitro*, resulting in "shedding" or continuous peeling off of surface macromolecules from non-dividing cells. The natural shedding of surface constituents into the serum may play an important role in the immune cellular reactivity of the host against its tumor. The dynamic nature of the cell surface is also manifested in its topography; when human and mouse cells are fused their surface antigens intermingle within 15

min, depending on temperature. The cell surface is continually forming transient structures, the microvilli, which project and retract, and probably play an important role in cell-to-cell interactions of both an immunological and a nonimmunological nature. (57 references)

- 3608 LEUKEMIA AND IRRADIATION. (E.) Montana, G. S. (U. North Carolina Sch. Med., Chapel Hill) and C. McMillan. *NC Med J* 34(10):785-788, 1973.

The dual relationship, causal and therapeutic, between irradiation and leukemia is discussed. The therapeutic value of irradiation in leukemia is well recognized. Irradiation is used in a variety of ways to palliate symptoms as well as, in rare instances, with the intent of eradicating this disease. Shrinkage of enlarged lymph nodes that cause pain, discomfort, or disfigurement, and relief of symptomatic bone involvement can be achieved with doses ranging from 600 rads in two days to 2,500 rads in two wk, depending on the location and extent of the involvement. Splenic irradiation has been found to be valuable in the treatment of chronic myelogenous leukemia, although caution is recommended since the white blood cell count can fall very rapidly. Whole body irradiation has resulted in clinical improvement in certain cases, while the most that can be expected of extracorporeal irradiation is a temporary reduction in the number of leukemic cells, and thus temporary remission of the leukemic process due to the rate of production of leukemic cells and other factors. Internal radiation emitters such as ^{32}P are also recognized for their possible therapeutic value. However, chronic lymphocytic leukemia has a long natural history, and it is debatable whether any form of treatment prolongs survival. Sequential use of various chemotherapeutic agents and irradiation may offer the potential of complete eradication of the disease. The authors' experiences with this approach are presented. On the other hand, irradiation has been observed to have leukemic potential in human beings. Leukemia has been induced in persons subjected to occupational or accidental irradiation or exposed to small doses administered in the treatment of benign diseases. Individuals who have received therapeutic doses of radiation to localized areas, such as patients treated for carcinoma of the cervix, have not shown an increase in the incidence of leukemia. Leukemia has also been induced experimentally by irradiation in animals. The carcinogenic mechanism involved in these cases is unclear. (33 references)

- 3609 (O)-FORMS OF RNA-CONTAINING ONCOGENIC VIRUSES AS A MODEL FOR LATENT INFECTION. (Rus.) Irlin, I. S. (N. F. Gamaleia Inst. Epidemiol. Microbiol., Moscow, USSR). *Vestn Akad Med Nauk SSSR* (2):28-33, 1973. (35 references)

- 3610 CAUSES OF LEUKEMIAS. (Fr.) Bernard, J. (No affiliation). *Recherche* 4(38):837-849, 1973. (7 references)

- 3611 ADVANCES IN RESEARCH ON THE ANALYSIS AND FORMATION OF N-NITROSO COMPOUNDS. (Rus.) Bogovskii, P. A. (Internat'l. Agency Cancer Res., Lyon, France) and E. A. Walker. *Vestn Akad Med SSSR* (3):40-45, 1973. (No references)

- 3612 A GENETIC MODEL OF THE HOST RANGE RESTRICTION OF MURINE LEUKEMIA VIRUSES: A REVIEW AND A HYPOTHESIS. (E.) Yoshikura, H. (Inst. Med. Sci., U. Tokyo, Japan). *Jap J Exp Med* 43(1):1-7, 1973. (30 references)

- 3613 ON CLASSIFICATION OF CHEMICAL CARCINOGENS ACCORDING TO THE MECHANISM OF THEIR ACTION. (Rus.) Neiman, I. M. (U.S.S.R. Acad. Med. Sci., Moscow). *Vopr Onkol* 19(8):76-80, 1973. (2 references)

- 3614 PRIMARY HEPATIC TUMORS IN INFANCY AND CHILDHOOD. (E.) Pollice, L. (Inst. Path. Anat., U. Bari, Italy). *Am J Clin Pathol* 60(4):512-521, 1973. (63 references)

- 3615 CHROMOSOME VARIABILITY AND CARCINOGENESIS. (Rus.) Pogosiants, E. E. (Inst. Exp. Clin. Oncol., Moscow, USSR). *Vestn Akad Med Nauk SSSR* (1):49-54, 1973. (31 references)

- 3616 METHODS AND APPLICATION OF THE ORGAN CULTURE TECHNIQUE IN CLINICAL AND EXPERIMENTAL CANCER RESEARCH. (Ger.) Matthias, M. (Central Inst. Cancer Res., E. German Acad. Sci., Berlin). *Arch Geschwulstforsch* 41(4):382-397, 1973. (150 references)

- 3617 THE BIOLOGICAL INDIVIDUALITY OF HUMAN TUMOURS. (Ger.) Tanneberger, St. (Central Inst. Cancer Res., Berlin, E. Germany), K. Rieche, G. Butschak, M. Görlich, E. Magdon, C.-N. Schremmer, E. Heise and D. Becker. *Arch Geschwulstforsch* 41(2):177-196, 1973. (123 references)

- 3618 IS NON-SPECIFIC ULCEROUS COLITIS A PRE-CANCEROUS CONDITION? (Rus.) Judin, I. Ju. (Inst. Exp. Clin. Oncology, Moscow, USSR) and S. N. Ginzburg. *Vopr Onkol* 19(8):49-54, 1973. (48 references)

- 3619 CONTROL AND PREVENTION OF OCCUPATIONAL CANCERS. (Ger.) Konetzke, G. W. (East German Central Inst. Occupational Med., Berlin), B. Bugyi and V. Peter. *Arch Geschwulstforsch* 41(4):398-403, 1973. (No references)

- 3620 MAMMALIAN EPOXIDE HYDRASES: INDUCIBLE ENZYMES CATALYSING THE INACTIVATION OF CARCINOGENIC AND CYTOTOXIC METABOLITES DERIVED FROM AROMATIC AND OLEFINIC COMPOUNDS. (E.) Oesch, F. (Bioctr., U. Basel, Switzerland). *Xenobiotica* 3(5):305-340, 1973. (164 references)

- 3621 ASPECTS OF TUMOR IMMUNOLOGY AND IMMUNOTHERAPY. (Ger.) Schmidt, C. G. (Essen U. Tumor Res. Clin., W. Germany). *Helv Chir Acta* 40(1/2):77-86, 1973. (No references)
- 3622 POSSIBILITIES OF CHEMICAL MODIFICATION OF MEMBRANOUS PROTEINS OF TUMOR CELLS CONSIDERING IMMUNOLOGICAL ASPECTS. (Ger.) Kruger, W. ("Manfred von Ardenne" Res. Inst., Dresden German Democ. Repub.). *Arch Geschwulstforsch* 42(1):58-72, 1973. (84 references)
- 3623 CYTOGENETICS OF CLONAL GROWTHS. (E.) Lejeune, J. (U. Paris, France). *N Eng J Med* 289(6):320-321, 1973. (5 references)
- 3624 LEUKAEMIA AND CANCER IN RELATION TO IMMUNOSUPPRESSIVE THERAPY. (E.) Videbaek, A. (Gentofte Hosp., Copenhagen, Denmark). *Scan J Haematol* 10(4):241-243, 1973. (25 references)
- 3625 ACTUAL PROBLEMS OF DIAGNOSIS, SPREAD AND TREATMENT OF CERVICAL CANCER AND ITS PREPHASES. (Ger.) Eschbach, W. (Central Inst. Cancer Res., East German Acad. Sci., Berlin), R. Huber and U. Bergmann. *Arch Geschwulstforsch* 41(4):336-358, 1973. (67 references)
- 3626 PHAEOCHROMOCYTOMA. A CASE REPORT AND REVIEW OF LITERATURE. (E.) Parkar, A. H. T. (Dept. Pediatrics, U. Nairobi, Kenya). *E Afr Med J* 50(6):286-293, 1973. (8 references)
- 3627 PREDICTIVE ONCOLOGY. (PART 1). (E.) Bierman, H. R. (Loma Linda U. Sch. Med., Calif.). *Int Surg* 58(10):683-692, 1973. (No references)
- 3628 CELL KINETICS OF TUMOUR GROWTH AND CANCER TREATMENT. (Fr.) Tubiana, M. (Gustave Roussy Inst., Villejuif, France) and E. P. Malaise. *Pathol Biol* 21(6):647-664, 1973. (No references)
- 3629 INFECTION WITH HERPES-SIMPLEX VIRUSES 1 AND 2 (SECOND OF THREE PARTS). (E.) Nahmias, A. J. (Emory U. Sch. Med., Atlanta, Ga.) and B. Roizman. *N Engl J Med* 289(14):719-725, 1973. (165 references)
- 3630 DEFINITION AND CLASSIFICATION OF ACUTE LEUKEMIA. (E.) Palmer, J. G. (U. North Carolina Sch. Med., Chapel Hill). *NC Med J* 34(9):702-706, 1973. (12 references)
- 3631 ELECTRON MICROSCOPY OF HUMAN TUMORS: A SHORT REVIEW. (E.) Erlandson, R. A. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.). *Clin Bull* 3(1):14-19, 1973. (47 references)
- 3632 ON THE BIOLOGICAL ALKYLATING AGENTS. (E.) Haddow, A. (Royal Cancer Hosp., London, England). *Perspect Biol Med* 16(4):503-524, 1973. (397 references)
- 3633 CHORIOANGIOMA OF THE PLACENTA. CASE REPORT. (E.) Sumathy, V. (Kansas City Gen. Hosp., Med. Ctr., Kansas), E. M. Grimes and G. L. Miller. *Mo Med* 70(9):647-649, 1973. (27 references)
- 3634 TRENDS IN HUMAN LEUKAEMIA. (E.) Anonymous. *Nature [New Biol]* 245 (140):1-2, 1973. (No references)
- 3635 PRIMARY BRAIN TUMORS: TUMOR IMMUNITY AND IMMUNOCOMPETENCE. (E.) Mavligit, G. M. (M.D. Anderson Hosp., Tumor Inst., Houston, Tex.), J. U. Gutterman and E. M. Hersh. *Surg Neurol* 1(5):261-263, 1973. (28 references)
- 3636 REVERSE TRANSCRIPTASE IN ACUTE LEUKAEMIA. (E.) Anonymous. *Lancet* (7828):542-544, 1973. (19 references)
- 3637 CARCINOEMBRYONIC ANTIGEN (CEA) AND GASTRO-INTESTINAL MALIGNANCY. (E.) Holyoke, E. D. (Roswell Park Inst., New York, N.Y.), T. M. Chu and G. P. Murphy. *Rev Surg* 30(5):305-311, 1973. (39 references)
- 3638 MALIGNANT LYMPHOMA OF THE LARYNX. (E.) Babbitt, D. C. (U. Nebraska Med. Ctr., Omaha), C. T. Yarrington, Jr. and A. J. Yonkers. *J Laryngol Otol* 87(8):807-810, 1973. (17 references)
- 3639 IMMUNOGLOBULINS ON THE SURFACE OF HUMAN LYMPHOCYTES. (E.) Aisenberg, A. C. (Harvard Med. Sch., Boston, Mass.). *Hum Pathol* 4(3):301-303, 1973. (16 references)
- 3640 THRESHOLDS AND LOW-LEVEL IRRADIATION EFFECTS. (E.) Anonymous. *Lancet* (7829):599-601, 1973. (16 references)
- 3641 EPIDEMIOLOGY OF HODGKIN'S DISEASE. (E.) Anonymous. *Lancet* (7830):647-648, 1973. (25 references)
- 3642 ORIGIN OF TERATOMAS. (E.) Ashley, D. J. B. (Morriston Hosp., Swansea, Wales). *Cancer* 32(2):390-394, 1973. (30 references)
- 3643 ANIMAL MODELS FOR AGING AND CANCER RESEARCH. (E.) Hollander, C. F. (Inst. Exp. Gerontology, Rijswijk, Netherlands). *J Natl Cancer Inst* 51(1):3-5, 1973. (18 references)

3644 THE COMBINED APPLICATION OF CYTOPHOTOMETRIC AND AUTORADIOGRAPHIC METHODS IN THE STUDY OF THE PROLIFERATION OF NORMAL AND DISTURBED HUMAN HAEMOPOIETIC CELL SYSTEMS. (Ger.) Queisser, W. (Fac. Clin. Med. Mannheim, U. Heidelberg, West Germany). *Klin Wochenschr* 51(14/15):687-694, 1973. (92 references)

3645 PEDIATRIC ONCOLOGY. (E.) Jaffe, N. (Harvard Med. Sch., Boston, Mass.). *Am J Med Technol* 39(9):345-353, 1973. (54 references)

- 3646 AFLATOXIN INHIBITION OF RAT LIVER MITOCHONDRIA. (E.) Doherty, W. P. (Dept. Biochem. Nutrition, Virginia Polytechnic Inst., Blacksburg) and T. C. Campbell. *Chem Biol Interact* 7(2):63-77, 1973.

When aflatoxin B₁ (AFB₁) is added to an actively respiring rat liver mitochondrial preparation, 25-44% inhibition of electron transport is produced with concentrations ranging from $2.5-4.8 \times 10^{-4}$ M, respectively. The degree of inhibition levels off at 4.8×10^{-4} M, which was shown to be in agreement with the critical micelle concentration. Submitochondrial or Gregg particles exhibit a maximum of 63% inhibition. Weanling rats maintained on a 5% casein semipurified diet for 15 days showed an approximate 30-50% reduction in the degree of aflatoxin inhibition for both mitochondria and Gregg particles compared to control animals fed a 20% casein diet *ad libitum*. The mitochondria of the protein-deprived animals had similar respiratory control ratios to normal animals. Dietary protein deficiency appears to exert its effect primarily at the site of action of aflatoxin rather than on alterations in membrane transport. The major site of inhibition of electron transport appeared to be between cytochromes b and c (c₁) as indicated by comparison of systems employing various substrates which donate their electrons to various portions of the electron transport system. At concentrations just below critical micelle formation, AFB₁ also reduced the ADP:O ratio, which was partially relieved by protein deficiency. These findings suggest that aflatoxin may function outside the cell nucleus. One possible consequence is that the likelihood of an interaction with mitochondria may be related to its necrogenic property.

- 3647 EFFECT OF BCG ON CARCINOGEN-INDUCED TUMOR DEVELOPMENT IN MICE. (E.) Kataoka, T. (Nat'l. Inst. Hlth., Tokyo, Japan), R. M. Nakamura, S. Yamamoto, T. Tokunaga, T. Murohashi and T. Tanaka. *Jap J Med Sci Biol* 25(6):377-382, 1972.

The effects of BCG inoculations before and after 20-methylcholanthrene (MCA) injection and those before and during oral administration of N-nitrosodiethylamine (DEN) were examined. In both cases 6-7 wk old female ddY mice were used. One hundred ninety mice were divided into two groups, one of which was sensitized with BCG. Four wk later, both groups were injected s.c. with 0.5 mg MCA. At 19 and 25 wk after MCA injection, the BCG sensitized mice were injected s.c. with 10^7 live BCG. The first tumors appeared in both groups in 12 wk, and the number of tumor-bearing animals increased wk by wk at almost equal rates. However, the difference in the cumulative incidence between the groups became gradually larger. In the other experiment, 224 mice were divided into two groups, one of which was sensitized with BCG. From six wk after BCG inoculation, all 224 mice were forced to drink water containing 50 ppm DEN for the first 11 wk and 25 ppm DEN for the next 10 wk. In the 19th wk, each BCG-sensitized mouse received another s.c. injection of 10^7 living BCG. A definite delay in development of tumors was observed in the BCG-sensitized mice, although the

final cumulative incidence was similar to that of the nonsensitized mice. These data indicate that BCG inoculations have some inhibitory effects on the development of tumors induced by the two carcinogens, MCA and DEN. It is suspected that this inhibition involves amplification of macrophage or reticuloendothelial function. The timing of BCG sensitization and the exposure to the carcinogen may be an important factor in these results.

- 3648 HISTOLOGIC AND ULTRASTRUCTURAL STUDIES ON THE HEPATOCARCINOGENICITY OF BENZENE HEXACHLORIDE IN MICE. (E.) Ito, N. (Nara Med. U., Japan), H. Nagasaki, M. Arai, S. Sugihara and S. Makiura. *J Natl Cancer Inst* 51(3):817-826, 1973.

Histologic and ultrastructural studies were made on hepatocarcinogenesis induced in male strain dd mice by the organochloride pesticide, benzene hexachloride (BHC). Mice were fed isomers of BHC for 24 weeks and then killed. Nodular hyperplasia and hepatocellular carcinoma were observed in mice fed basal diet containing 500 parts per million (ppm) of the α -isomer of BHC (α -BHC) (100% incidence), 250 ppm α -BHC (79%), 250 ppm α -BHC plus 250 ppm β -BHC (93%), 250 ppm α -BHC plus 250 ppm γ -BHC (93%), or 250 ppm α -BHC plus 250 ppm δ -BHC (75%). However, diet containing other isomers, or combinations of 100 ppm α -BHC and these isomers, did not induce liver tumors. Histologically, liver nodules induced in mice by α -BHC were well-differentiated hepatocellular carcinomas or nodular hyperplasia. Cirrhosis was rare. Electron microscopic examination showed a marked increase in the smooth endoplasmic reticulum in the cytoplasm of cells in areas of hepatocellular carcinoma, with frequent nuclear and mitochondrial changes. Liver tumors were induced in mice only by α -BHC, whereas β -, γ -, and δ -BHC had no synergistic or antagonistic effects on the induction of tumors by α -BHC.

- 3649 CELLULOSE DIETARY BULK AND AZOXYMETHANE-INDUCED INTESTINAL CANCER. (E.) Ward, J. M. (Nat'l. Cancer Inst., Bethesda, Md.), R. S. Yamamoto and J. H. Weisburger. *J Natl Cancer Inst* 51(2):713-715, 1973.

A model experiment explored the significance of bulk in colon cancer production. Male Fischer rats were fed a low-residue semisynthetic diet or the same diet containing 20% and 40% of its bulk as cellulose. All animals received azoxymethane (14.8 mg/kg, s.c.) once a wk for 10 wk and were held an additional 16 wk. Daily food intake and fecal wt were proportional to the inert cellulose content of the diet. Most animals inoculated with the colon carcinogen had tumors in the intestinal tract. However, tumors in the small intestine were fewer than the average number of all intestinal tumors/rat, with the highest number on the semisynthetic diet and progressively lower numbers as the bulk of the diet increased. In the colon there were slightly more tumors/rat when the diet contained 20% cellulose. Thus azoxymethane-induced tumors in the small intestine were more sensitive to bulk and fiber content of the diet than were tumors in the colon.

- 3650 CALCIUM-DEPENDENT STIMULATION BY A PHORBOL ESTER (PMA) OF THYMIC LYMPHOBLAST DNA SYNTHESIS AND PROLIFERATION. (E.) Whitfield, J. F. (Dept. Biol. Sci., Natl Res. Council Canada, Ottawa), J. P. MacManus and D. J. Gillan. *J Cell Physiol* 82(2):151-156, 1973.

Thymic lymphocyte populations isolated from male albino rats were suspended in MAC-1 medium containing 0.5 mM calcium and exposed to several concentrations of phorbol myristate acetate (PMA), i.e., 12-O-tetradecanoyl-phorbol-13-acetate, a tumor-promoting ester from croton oil. PMA at its most effective concentration of 0.05 µg/ml rapidly (within 1 hr) induced a large fraction of the lymphoblasts in the thymic cell populations to start making DNA. Afterwards, the stimulated cells rapidly left the S phase and progressed into mitosis. The stimulatory action of PMA is probably mediated by calcium because it disappeared when calcium was omitted from the medium. When the medium contained 0.05 µg/ml PMA, even a calcium concentration of 0.2 mM stimulated DNA synthesis.

- 3651 LYSOSOMAL ENZYMES IN 7,12-DIMETHYLBENZ[A] ANTHRACENE-INDUCED MAMMARY CARCINOMAS AFTER OVARECTOMY. (E.) Nicholson, R. I. (Welsh Natl. Sch. Med., Cardiff, Wales), I. Bagnall and M. Davies. *Eur J Cancer* 9(4):313-318, 1973.

Mammary tumors were induced in Sprague-Dawley rats by intubation feeding of a solution containing 20 mg dimethylbenz(a)anthracene in 1 ml sesame oil at age 50 days. The activities of the five acid hydrolases together with protein, DNA, and RNA were established in growing tumors and in tumors five days after ovariectomy. Although variations were recorded between the individual enzymes, the general trend was an increase in the specific activity of β -glycerophosphatase, *p*-nitrophenylphosphatase, acid proteinase, *N*-acetylglucosaminidase, and β -glucuronidase. The most marked increase in activity (4166 to 40,16 U/g protein) was recorded with acid proteinase, an important enzyme involved in the breakdown of tissue components. The results indicate that the pattern observed for the regressing tumor is similar to that observed for normal hormone-dependent tissue undergoing physiological changes.

- 3652 THE COMBINED EFFECT OF RADIATION AND CHEMICAL CARCINOGENS IN FEMALE A x IF MICE. (E.) Flaks, A. (Sch. Med., U. Leeds, England), J. M. Hamilton, D. B. Clayson and P. R. J. Burch. *Br J Cancer* 28(3):227-231, 1973.

Studies were conducted in female A x IF mice to determine the short and long-term effects of whole and lower body x-irradiation on the carcinogenic activity of the two known bladder carcinogens, dibutyl nitrosamine (DBNA) and 4-ethylsulphonylnaphthalene-1-sulphonamide (ENS). Animals received either a single dose of 500 or 100 rads or irradiation followed by s.c. DBNA or oral ENS treatment beginning 24 hr later. Postmortem gross and histological examinations showed no evidence of

bladder lesions in control animals treated with irradiation alone. Irradiation also had no influence on the incidence or latent period for the development of DBNA or ENS-induced bladder carcinomas. Epithelial hyperplasia and hemangioendotheliomas were also common in carcinogen-treated mice. Many treated animals developed urinary tract hydronephrosis secondary to calculus (ENS) or thrombus (DBNA) formation. Although mammary tumors were found in all groups, including controls, they were seen with increasing frequency in irradiated or carcinogen-treated mice. Combined treatment of 100 rad and ENS or DBNA markedly increased the incidence of mammary tumors compared to that of carcinogen-treated animals. Pulmonary tumors were also seen in all groups but were most frequent in DBNA treated mice.

- 3653 CAFFEINE INHIBITION OF POSTREPLICATION REPAIR OF *N*-ACETOXY-2-ACETYLAMINOFLUORENE-DAMAGED DNA IN CHINESE HAMSTER CELLS. (E.) Trosko, J. E. (Dept. Human Development, Michigan State U., East Lansing), P. Frank, E. H. Y. Chu and J. E. Becker. *Cancer Res* 33(10):2444-2449, 1973.

The effect of caffeine on Chinese hamster cells *in vitro*, treated with various metabolites and derivatives of 2-acetylaminofluorene, was studied at the molecular level. With the use of an alkaline sucrose gradient centrifugation technique, parental and newly synthesized DNA in control and treated cells were studied in the presence and absence of caffeine. Caffeine synergistically affected only the sedimentation profiles of DNA synthesized in *N*-acetoxy-2-acetylaminofluorene-treated cells but not in the control cells or in cells treated with the various derivatives of 2-acetylaminofluorene. *N*-Acetoxy-2-acetylaminofluorene also affected the sedimentation profiles of parental DNA, but caffeine did not influence this effect. At the dose level used, caffeine had no apparent effect on the incorporation of thymidine into DNA in either the control or *N*-acetoxy-2-acetylaminofluorene-treated cells. These results supplement other reports that suggest that the *N*-acetoxy-2-acetylaminofluorene lesion in DNA of either human or Chinese hamster cells is repaired similarly to UV light-induced pyrimidine dimers.

- 3654 THE METABOLISM OF ^{14}C AFLATOXINS IN LAYING HENS. (E.) Sawhney, D. S. (Dept. Poultry Sci., Cornell U., Ithaca, N.Y.), D. V. Vadehra and R. C. Baker. *Poult Sci* 52(4):1302-1309, 1973.

Sodium acetate- ^{14}C labeled aflatoxins were produced by growing *Aspergillus flavus* strain NRRL-2999 on rice. A single 0.29 µ Ci. oral dose of aflatoxins was administered to laying White Leghorn hens. The radioactivity distribution and its equivalents in various tissues, at one, four and seven days after the administration of the dose were determined. Seven days after treatment, 70.61% of the dose was recovered in the excrement. The excretion of aflatoxins or their metabolites into the intestine via the bile seemed to be the major pathway by which absorbed aflatoxins were excreted. All the components

of eggs laid at various intervals showed ^{14}C activity. Edible parts of the carcass showed varied amounts of ^{14}C aflatoxins and/or their metabolites at all the periods studied. The time necessary to eliminate one-half of the radioactive aflatoxins from the body was found to be 66.82 hr. The liver, crop, gizzard and fecal material when fed in the diets were toxic to the duckling.

- 3655 STUDIES ON SERYL AND PHENYL-ALANYL SYNTHETASES FROM ASCITIC TUMOR CELLS TREATED WITH 20-METHYLCHOLANTHRENE. (E.) Pedersen, K. (Fibiger Lab., Copenhagen, Denmark) and R. Quist. *Proc Soc Exp Biol Med* 144(2):714-721, 1973.

Phenylalanyl and seryl synthetase activities were studied in C3H-L1a ascites tumor cells treated *in vitro* with 20-methylcholanthrene and harvested during their exponential growth phase 3-4 days after i.p. inoculation into inbred C3H mice. The two enzymes were purified 400- to 600-fold from cell homogenates by DEAE cellulose, CM Sephadex C-50, and hydroxyapatite column chromatography. Enzyme activities were determined by the extent of ^{14}C -labeled amino acid fixed to purified rat liver tRNA. Kinetic studies showed that the K_m for serine was about 20 times higher in MCA-treated cells than in controls, whereas the K_m for ATP was increased only three- to four-fold. These results indicated that the synthetases probably have a regulatory role in protein synthesis of cancer cells with and without treatment with MCA. Similar differences between MCA-treated cells and controls with respect to the K_m for phenylalanyl transferase were not observed.

- 3656 TUMOUR METASTASIS IN MICE WITH REDUCED IMMUNE REACTIVITY. I. STUDIES WITH TWO MCA-INDUCED SARCOMAS IN RADIATION AND THYMECTOMIZED RADIATION C57BL/6J CHIMERAS. (E.) Boeryd, B. (Dept. Path. I, U. Göteborg, Sweden) and M. Suurküla. *Int J Cancer* 12(3):722-727, 1973.

Spontaneous tumor metastasis was studied in 3-month-old male and female x-irradiated or thymectomized and x-irradiated C57BL/6J mouse chimeras bearing one of two methylcholanthrene induced sarcomas differing in antigenicity. Animals were immunized by s.c. tail implantation followed in 2 wk by tail excision. Five days later, the animals were challenged with graded doses of a suspension of dissociated tumor cells. Challenge of mice immunized with one tumor cell type by s.c. inoculation with the second tumor cell type showed that no cross-reactivity existed between the two. Thymectomized mice showed no enhancement of immune reactivity when compared with controls. Spontaneous metastasis formation was not influenced in either of the tumors by immune suppressive treatment, although tumor growth was decreased in all irradiated groups compared with controls except for the thymectomized chimeras. Possible explanations for these findings are that the immune factors have no influence on metastasis formation in these two

systems or that immune suppressive treatment does not influence interplay between postulated facilitating and inhibiting factors.

- 3657 INFLUENCE OF PREINFECTION OF C57BL/6 MICE WITH GRAFFI LEUKEMIA VIRUS ON 3-METHYLCHOLANTHRENE-INDUCED SUBCUTANEOUS SARCOMA. (E.) Whitmire, C. E. (Microbiol. Assoc., Bethesda, Md.), and R. A. Salerno. *Proc Soc Exp Biol Med* 144(2):674-679, 1973.

Sarcoma formation following s.c. injection of 3-methylcholanthrene (MCA) was studied in male and female C57BL/6 mice previously inoculated i.p. with Graffi type C RNA virus, a virus which induces lymphatic leukemia. The incidences of lymphoma and sarcoma were dependent on the dose of virus or MCA given. Inoculation with high virus titers ($10^{-1.5}$) reduced the incidence of MCA-induced sarcomas since these mice usually died of leukemia before the mean latent period for sarcoma formation had passed. High doses (300 μg) of MCA increased the incidence of leukemia induction by low virus titers ($10^{-2.5}$) without affecting the incidence of sarcoma. This occurred since the latency period for sarcoma and leukemia coincided and a significant fraction (25%) of the mice had both sarcomas and leukemia at the time of autopsy. Combination of a low virus titer and low dose of MCA (25 or 100 μg) produced no change in the incidence of leukemia or sarcoma. Under these circumstances, however, the mean latent period for leukemia development was prolonged and that for sarcoma formation was decreased.

- 3658 TYPE OF ASBESTOS AND RESPIRATORY CANCER IN THE ASBESTOS INDUSTRY. (E.) Enterline, P. E. (Graduate Sch. Public Hlth., U. Pittsburgh, Pa.) and V. Henderson. *Arch Environ Health* 27(5):312-317, 1973.

The mortality experience of 1,348 retired asbestos workers was studied in an attempt to correlate the incidence of respiratory cancer with the type of asbestos to which they were exposed. The range of exposure of these men was 30 to 51 yr within an average of 25 yr. Historic dust levels were in excess of 50 million particles per cubic foot of air. Of 754 deaths observed between 1941 and 1967, death certificates were available for 733. The cohort showed an overall mortality rate 15.1% higher than the general white US male age-matched population which was almost entirely due to cancer and respiratory diseases. This group showed greatest excesses of respiratory cancer, pneumoconioses and pulmonary fibrosis. The respiratory cancer excess was about the same for each age group studied. Although the individual groups of men exposed to the different types of asbestos were small and the results, therefore, inconclusive, the data suggested that crocidolite asbestos might be more carcinogenic than either amosite or chrysotile. Separate study of retired cement industry workers showed that men exposed to crocidolite plus chrysotile had a respiratory cancer risk 6.1 times the expected compared

to only a 1.4 times greater risk for men exposed only to chrysotile.

- 3659 MALIGNANT TRANSFORMATION *IN VITRO* OF RAT LIVER CELLS BY DIMETHYLNITROSAMINE AND N-METHYL-N'-NITRO-N-NITROGUANIDINE. (E.) Montesano, R. (Internatl. Agency Res. Cancer, Lyon, France), L. Saint Vincent and L. Tomatis. *Br J Cancer* 28(3):215-220, 1973.

The transforming abilities of dimethylnitrosamine (DMN), an indirect alkylating agent requiring metabolic activity, and of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), a direct alkylating agent, were studied in epithelioid cells subcultured from the livers of eight- and ten-day-old BD VI rats. Cells treated for four wk with MNNG or for one wk with DMN showed no morphological change compared to untreated controls; however, such cells acquired the ability to form growing colonies in soft agar. S.C. injection of MNNG transformed cells resulted in carcinosarcoma formation after three months in 9 of 10 newborn rats. I.P. injection of DMN transformed cells into newborn rats resulted in ascites and the formation of adenocarcinomas after 9 to 12 wk in all six animals studied. Both tumor types metastasized readily. These results indicated that *in vitro* transformed epithelioid cells do not necessarily produce carcinomas when injected into suitable hosts. Such cell lines may also contain mesenchymal cells which may also be transformed.

- 3660 URETHAN-INDUCED PULMONARY ADENOMA AS A TOOL FOR THE STUDY OF SURFACTANT BIOSYNTHESIS. (E.) Snyder, C. (Med. Div. Oak Ridge Assoc. U., Tenn.), B. Malone, P. Nettesheim and F. Snyder. *Cancer Res* 33(10):2437-2443, 1973.

Pulmonary adenomas induced in female BALB/c mice by urethan 1 mg/g, i.p., on three consecutive days contain significant quantities of disaturated phosphatidylcholine, the main constituent of lung surfactant. Data obtained after phospholipase C hydrolysis, acetate derivatization of the resulting diacylglycerols, and then argentation thin-layer chromatography indicated that the disaturated molecular species represent about 28% of the phosphatidylcholine present in the adenoma cells and that at least 60% of this fraction is 1,2-dipalmitoyl-*sn*-glycero-3-phosphorylcholine. Homogenate preparations, in the presence of added adenosine triphosphate, coenzyme A and Mg^{2+} , incorporated palmitate at positions 1 and 2 of the glycerol moiety, suggesting that the alveolar type II epithelial cells that comprise the pulmonary adenoma contain an enzymic system capable of producing disaturated phosphatidylcholine. Adenoma homogenates also produce labeled alkyl glycerolipids from hexadecanol-1- 3H . This is characteristic of most tumors, including the lung squamous cell carcinoma that was tested. The carcinoma also actively incorporated palmitic acid into phosphatidylcholine but not to the same extent that the adenoma did. The addition of Ca^{2+} did not inhibit the incorporation of palmitic acid-1- ^{14}C into phosphatidylcholine but decreased its total incorporation into lipids. In contrast, the

formation of *O*-alkyl phospholipids from hexadecanol-1- 3H was completely blocked by Ca^{2+} . Since Ca^{2+} is known to inhibit cytidine diphosphate-base transferase activity, these data demonstrate that the major incorporation of palmitic acid-1- ^{14}C into phosphatidylcholine in the adenoma homogenate does not take place via the cytidine diphosphate choline pathway or by methylation of phosphatidylethanolamine.

- 3661 CARCINOGENICITY OF PHENOLS, ALKYLATING AGENTS, URETHAN, AND A CIGARETTE-SMOKE FRACTION IN *NICOTIANA* SEEDLINGS. (E.) Andersen, R. A. (Dept. Agronomy, U. Kentucky, Lexington). *Cancer Res* 33(10):2450-2455, 1973.

Aqueous solutions of the phenols pyrogallol, resorcinol, and 3-hydroxyanthranilic acid, several alkylating agents (*N*-nitrosamines and β -propiolactone), urethan, nucleic acid analogs (6-azauracil and 6-azauridine), and a water-soluble extract of cigarette-smoke condensate induced tumors during early seedling development in one or both of two higher plants. The seeds of tumor-prone interspecific hybrids of the diploid *Nicotiana glauca* X *Nicotiana langsdorffii* and the tetraploid *Nicotiana suaveolens* X *N. langsdorffii* were soaked in aqueous solutions of these agents for 24-48 hr, and tumors were scored on germinated seedlings 20 days later. The carcinogenic activity of several of the active compounds or structurally related compounds has already been demonstrated in animal bioassay systems. The parallel activity in *Nicotiana* hybrids suggests a mechanism of oncogenic action common to certain plant and animal tissues. 6-Azauracil was the most potent carcinogen tested in both hybrids at 0.01-0.1 mM and was also the most active at 1 mM. The next most active compound was pyrogallol (0.1 and 1 mM).

- 3662 MAMMARY NEOPLASIA IN THE FEMALE RAT AFTER NEONATAL AND/OR ADULT 7,12-DIMETHYLBENZ-(a)ANTHRACENE ADMINISTRATION. (E.) Brown, R. D. (Natl. Inst. Arthritis Metabolism Digestive Dis., Bethesda, Md.), A. R. Rao and C. J. Shellabarger. *Proc Soc Exp Biol Med* 144(1):96-98, 1973.

Possible interactions of two doses of 7,12-dimethylbenz(a)anthracene (DMBA), one given to neonatal female Sprague-Dawley rats and the other given to young, adult females were investigated. It was hoped to learn if the young, adult female Sprague-Dawley rats given DMBA would demonstrate a "memory response" to a previous, neonatal administration of DMBA. At 175 days of age it was found that 0.3 mg DMBA in 0.05 ml sesame oil given to the rats at two days of age was followed by a high incidence of mammary fibroadenoma. DMBA (13.3 mg/100 g body weight) given at 52 days of age was followed by a high incidence of mammary adenocarcinoma. When both DMBA treatments were given to the same animals, the mammary adenofibroma incidence was much the same as in the rats given only DMBA at 2 days of age and mammary adenocarcinoma incidence was much the same as in animals given only DMBA at 52 days of age. No significant interaction between two doses of DMBA

given at both 2 and 52 days of age on mammary neoplasia was noted.

- 3663 ALTERATIONS IN THE LDH ISOENZYMES OF RAT BRONCHIAL MUCOSA DURING CHEMICAL CARCINOGENESIS. (E.) Turner, D. M. (Tobacco Res. Council Labs., Harrogate, England), B. R. Davis and H. L. Crabb. *Enzyme* 14(3):166-172, 1973.

Lactate dehydrogenase isoenzymes were studied in the bronchial mucosa of 96 female SPF Wistar rats treated, by intratracheal intubation, with 2 mg 3,4-benzo(a)pyrene (BP) every 14 days for 58 wk. After 26 wk (14 treatments), the isoenzymes showed a shift toward the electrophoretically slower moving zones, relative to controls, in the group of animals treated with 2 mg BP. Animals treated with 1 mg BP at the same intervals did not show a significant change in isoenzyme pattern until 42 wk (22 treatments). Many mucosal patterns, from animals treated with BP, showed a pattern shift in the absence of any microscopically detectable changes in the lungs. The shift in pattern was as marked in those animals with squamous metaplasia as in those with carcinoma although a dose response relationship may be seen in regard to the numbers of tumors produced by the BP.

- 3664 INDUCTION OF LIVER AND LUNG TUMOURS IN RATS BY THE SIMULTANEOUS ADMINISTRATION OF SODIUM NITRITE AND MORPHOLINE. (E.) Newberne, P. M. (Dept. Nutrition Food Sci., Massachusetts Inst. Technol., Cambridge) and R. C. Shank. *Food Cosmet Toxicol* 11(5):819-825, 1973.

The incidence of liver and lung tumors was determined in female Sprague-Dawley rats fed a diet containing various concentrations (up to 1000 ppm each) of nitrite and morpholine. Almost 100% of 159 rats fed a diet with 1000 ppm of each substance developed hepatocellular carcinoma and about 25% of these had coexisting hepatic angiosarcomas which appeared to arise from either sinusoidal endothelium or Kupffer cells. Over 68% of the animals with hepatic tumors had pulmonary metastases. Decreasing either nitrite or morpholine to less than 50 ppm while maintaining the other substance at 1000 ppm resulted in a decrease in tumor incidence to less than 4% with no animals showing metastatic disease. The tumors induced by nitrite plus morpholine were histologically identical to those induced by oral N-nitrosomorpholine administration. These results supported the concept that *in vivo* nitrosation of morpholine does occur in the rat, presumably in an acidic gastric environment.

- 3665 THE EFFECT OF pH ON DIMETHYLNITROSAMINE FORMATION IN HUMAN GASTRIC JUICE. (E.) Lane, R. P. (Dept. Food Sci. Nutrition, U. Missouri, Columbia) and M. E. Bailey. *Food Cosmet Toxicol* 11(5):851-854, 1973.

The formation of dimethylnitrosamine (DMNA) from 100 ppm nitrite and 100 ppm dimethylamine was

studied in a 50% aqueous solution of human gastric juice over a pH range of 1.7 to 4.5. The reaction product was purified by steam distillation from alkaline solution followed by methylene chloride extraction and Celite column chromatography, and was identified by gas-liquid chromatography and high resolution mass spectrometry. DMNA was formed over the entire pH range and tested in amounts ranging from 9 to 34 parts per billion. Preliminary results indicated a pH optimum of 2.5 for the reaction, rather than the 3.4 previously found in aqueous solutions free of HCl. Quantitative analysis of several pooled gastric juice samples showed the presence of 1 mg thiocyanate/100 ml which was thought to be responsible for the shift in the pH optimum. These data suggested that reaction mechanisms involving nitrosyl chloride and/or nitrosyl thiocyanate intermediates may be involved in the nitrosation of dimethylamine by human gastric juice.

- 3666 POSSIBLE ASSOCIATION BETWEEN BENIGN HEPATOMAS AND ORAL CONTRACEPTIVES. (E.) Baum, J. K. (U. Michigan Hosp., Ann Arbor), F. Holtz, J. J. Bookstein and E. W. Klein. *Lancet* (7835):926-929, 1973.

Primary benign hepatomas were discovered and confirmed histologically in seven young southern Michigan women on oral contraceptives within the past 5 yr. Five of the seven, who ranged in age from 25 to 39, presented with acute abdomens. The remaining two had right upper quadrant masses. Massive i.p. hemorrhage was the most common finding. Coagulation disorders secondary to hepatocellular disease were not uncommon. All five of the patients in which the type of oral contraceptive was known had been on a combination type for six months to seven yr. Hepatic arteriography provided a prompt and accurate diagnosis.

- 3667 CARCINOGENICITY OF CHOLESTEROL INHIBITED BY PHOSPHOLIPIDS. (E.) Altman, R. F. A. (Inst. Oswaldo Cruz, Rio de Janeiro, Brazil). *Arch Geschwulstforsch* 41(2):107-109, 1973.

The effect of simultaneous administration of phosphatidylcholine on the cholesterol-induced development of fibrosarcoma was studied in adult Swiss mice. Experimental animals were fed either a normal diet or one containing lard (80 ml/kg body wt.) In addition, all animals received three s.c. injections of 10% cholesterol in olive oil at 3 wk intervals. One group also received three s.c. injections of 20% purified phosphatidylcholine and 1% soybean phosphatide (Asolectin) in the drinking water. Animals were examined twice weekly for tumors. Whereas no tumors were observed in the controls, tumors began to develop in the experimental mice 12 wk after the final cholesterol injection. After 50 wk, overall mortality was 67.5% and 57.5% (of 40 animals) for mice receiving s.c. cholesterol and s.c. plus oral cholesterol, resp. Overall mortality of the mice which also received s.c. plus oral phospholipid was, however, only 17.5%. These results indicated that simultaneous administration of phosphatidylcholine strongly in-

hibited cholesterol-induced tumorigenesis in Swiss mice.

- 3668 EFFECTS OF THE CARCINOGEN N-ACETOXY-2-FLUORENYLACETAMIDE ON THE TEMPLATE PROPERTIES OF DEOXYRIBONUCLEIC ACID. (E.) Zieve, F. J. (Minneapolis Veterans Hosp., Minn.). *Mol Pharmacol* 9(5):658-669, 1973.

Treatment of DNA *in vitro* with the carcinogen N-acetoxy-2-fluorenylacetamide (N-acetoxy-2-FAA) drastically reduced the capacity of the DNA to serve as a template for RNA synthesis. Significant inactivation of template activity occurred within seconds after mixing the carcinogen with DNA. A given quantity of treated DNA could bind 10 times more RNA polymerase than the same quantity of control DNA. RNA synthesis directed by treated DNA ceased after 5 min of incubation, while RNA synthesis directed by control DNA continued for over 1 hr. Treated DNA was as effective as control DNA in supporting the exchange of [32 P]pyrophosphate into nucleoside triphosphates in the presence of RNA polymerase. These findings indicated that N-acetoxy-2-FAA inhibited RNA synthesis by preventing chain elongation, and that chain initiation was unaffected. Denaturation by heat or alkali decreased the template capacity of control DNA but increased the template capacity of treated DNA. In addition, treated DNA was more effective than control DNA in supporting the homopolymerization of adenylic acid by RNA polymerase. It therefore appeared likely that N-acetoxy-2-FAA produced regions of partial denaturation of DNA. Comparison of the effects of a series of N-acetoxyarylacetamides revealed that abolition of DNA template activity only occurred when the aryl moiety was greater than one ring in size and when the nitrogen atom was located *para* to the aromatic system.

- 3669 EFFECTS OF HYPOTHYROIDISM AND PROGESTERONE ON MAMMARY TUMOURS INDUCED BY 7,12-DIMETHYLBENZ(a)ANTHRACENE IN SPRAGUE-DAWLEY RATS. (E.) Jabara, A. G. (Dept. Path. Statistics, U. Melbourne, Australia) and J. S. Maritz. *Br J Cancer* 28(2):161-172, 1973.

Female Sprague-Dawley rats were injected i.p. with 1 mCi 131 I as iodide in thiosulfate solution at age 30 days. Hypothyroidism, alone or combined with progesterone (3 mg, s.c., twice a wk for 28 wk), significantly decreased 7,12-dimethylbenz(a)anthracene (DMBA, 30 mg, intragastrically) mammary tumorigenesis relative to controls. However, the decrease was less in the progesterone-treated group, and statistical analysis showed that progesterone enhanced tumorigenesis to the same extent in hypothyroid animals as in the controls. Most tumors in hypothyroid progesterone-treated rats were adenocarcinomas; in the absence of the hormone most tumors were benign. However, the difference between the tumor types in the two groups was not statistically significant. The morphological changes observed in the endocrine glands, genital tracts and non-neoplastic mammary tissue, considered in relation to previously reported data, suggest that hypothyroidism

reduced the tumor yield mainly by secondarily inhibiting somatotrophin production and secretion, although the effect of decreased food intake could not be excluded completely. The higher tumor yield in the hypothyroid progesterone-treated rats may have been due to higher circulating levels of prolactin in this group compared with those in the hypothyroid group which received no hormone.

- 3670 THE REACTION OF 14 C-LABELLED PLATINUM ETHYLENEDIAMINE DICHLORIDE WITH ADENINE COMPOUNDS AND DNA. (E.) Robins, A. B. (Inst. Cancer Res., Belmont, Sutton, Surrey, England). *Chem Biol Interact* 7(1):11-16, 1973.

Various derivatives of adenine have been studied with regard to their rate of reaction with 14 C-labeled platinum ethylenediamine dichloride, $Pt(^{14}C-en)Cl_2$. The reactivities have been calculated from the "rate of disappearance" of $Pt(^{14}C-en)Cl_2$ using chromatographic separation of reactants and products. Adenine and adenosine react very slowly at 37 C whereas other adenine derivatives react much more readily in the order: poly A > AMP > ApA > poly d(AT). From the numerical values of the rate constants it is concluded that the presence of a phosphate group increases the reaction rate considerably. This is partly the explanation of the rapid reaction of poly A which possesses terminal phosphate groups. However adjacent adenine moieties such as those in poly A and ApA may also react by another mechanism which involves the 6-NH₂ groups. In DNA, no free phosphate groups are present, and the occurrence of adjacent adenines will be low. The reaction of $PtenCl_2$ with DNA seems to involve a rapid attack on deoxyguanosine and a slow reaction with deoxyadenosine and deoxycytidine.

- 3671 METHYLGUANIDINE, A NATURALLY OCCURRING COMPOUND SHOWING MUTAGENICITY AFTER NITROSATION IN GASTRIC JUICE. (E.) Endo, H. (Fac. Med., Kyushu U., Fukuoka, Japan) and K. Takahashi. *Nature* 245(5424):325-326, 1973.

N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), which induced a high frequency of tumors in the glandular region of the rodent and dog stomach on oral application, is synthesized by nitrosation of N-methyl-N'-nitroguanidine (MNG) not only in strongly acidic conditions but also in human gastric juice. The mutagenicity of the 14 naturally occurring guanidines, including methylguanidine (MG), was examined after treatment with nitrite in real and in simulated human gastric juice. Deep rough derivatives of histidine-requiring strains of *Salmonella typhimurium* carrying the *gal-bio-uvr B* deletion were used for the mutagenicity assay. Samples of a guanidine derivative to be tested, sodium nitrite and hydrochloric acid in a molar ratio of 1:1:1.3 were spotted onto lawns of the tester strains of bacteria plated on minimal agar. Following two days incubation at 37 C revertants were observed. His⁺ revertants of the TA1535 strain appeared because of production of a mutagen causing base-pair substitutions in the reaction mixture. Nitrosated MG was found to be mutagenic at up to 16-fold dilution of the original reaction mixture; all other guanidines tested failed to show mutagenicity

detectable by this spot test. MG is structurally quite similar to MNG and is found in fresh beef, ray, cod, sardines and especially in sharks, at quite high concentrations (1,900 mg/kg fresh wt). It therefore seems possible that nitrosated MG is one of the causes of "spontaneous" human gastric cancers.

- 3672 OXIDATION OF CARCINOGENIC AZO-DYES XIII. METABOLITES OF SOME 3'-SUBSTITUTED DERIVATIVES OF DIMETHYLAMINOAZOBENZENE IN THE BILE OF RATS. (E.) Marhold, J. (Res. Inst. Organic Synthesis, Pardubice-Rybitvi, Czechoslovakia), V. Rambousek, J. Pipalova, J. Kroupa and M. Matrká. *Neoplasma* 20(1):27-30, 1973.

The metabolites of nine derivatives of N,N-dimethyl-4-aminoazobenzene substituted in position 3' were determined chromatographically in the bile of rats after their p.o. administration, (0.5 g/kg). 4'-hydroxylated metabolites occurred after the administration of F, Cl, Br, CH₃, and OCH₃ derivatives in the position 3', whereas no such metabolites were detected after the administration of I, COOH, NO₂, and NHCOCH₃ derivatives in the meta-position. In all cases, N-demethylated metabolites were demonstrated, including the derivatives of aminoazobenzene, which are not products of any oxidation *in vitro*. Both 4'-hydroxylation and N-demethylation occur in all proved strong carcinogens of the 3'-substituted series.

- 3673 MESOTHELIOMATA IN RATS AFTER INOCULATION WITH ASBESTOS AND OTHER MATERIALS. (E.)

Wagner, J. C. (Llandough Hosp., Penarth, Wales), G. Berry and V. Timbrell. *Br J Cancer* 28(2):173-185, 1973.

Four experiments in which SPF Wistar rats were inoculated intrapleurally with asbestos chrysotiles, crocidolite, brucite, barium sulphate, ceramic fiber, fiberglass, glass powder, aluminum oxide or standard reference samples from the International Union Against Cancer are described. Saline was used as a control. Mesotheliomas were observed in a considerable proportion of animals with all the samples of asbestos used and with a sample of brucite. A few were produced with synthetic aluminum silicate fibers and single ones with barium sulphate, glass powder and aluminum oxide. The risk of developing a mesothelioma at a given time after injection was approximately proportional to the dose. Of the UICC standard reference samples, crocidolite was the most carcinogenic and removal of the oils by benzene extraction did not alter the carcinogenicity of these samples. Chemical properties also seem unlikely to be the main factor producing mesotheliomas but the results support the hypothesis that the finer fibers are the more carcinogenic, and this is additional to the known aerodynamic advantage which the finer fibers have in penetrating to the periphery of the lung. The application of the test materials by intrapleural inoculation may be criticized as unrealistic in comparison with human exposure, about which the animal experiments are intended to provide relevant information. Inoculation experiments, however, are simpler to interpret than inhalation experiments since the varying penetration

of different samples of dust through the airways and alveoli is not a factor.

- 3674 HISTOPATHOLOGICAL STUDIES ON RAT COLORECTAL CARCINOMA INDUCED BY N-METHYL-N'-NITRO-N-NITROSOGUANIDINE. (E.) Nakano, H. (Tohoku U. Sch. Med., Japan). *Tohoku J Exp Med* 110(1):7-21, 1973.

Donryu rats received an infusion of various amounts of N-methyl-N'-nitro-N-nitrosoguanidine from the anus, were kept under observation for a maximum period of 61 wk, and sacrificed at certain intervals for histopathological studies. A total of 133 tumors was found in 59 out of 138 animals. These tumors were histologically: 19 adenocarcinomas, 73 adenomatous lesions, 20 hyperplastic lesions, and 21 hyperplastic lymph nodes. These lesions were found only in that segment of colon that the carcinogen contacted directly. These were all macroscopically elevated types, which included thickened folds. Tumor occurrence in the animals given 0.5 ml of 0.25% N-methyl-N'-nitro-N-nitrosoguanidine solution daily for 32 days (total dose of the carcinogen 40 mg), was 76% at the end of 40 wk after anal infusion. Adenomatous lesions were found at the end of 17 wk or later after infusion, while adenocarcinomas occurred at 35 wk. Histologically, all the lesions of adenocarcinoma were located immediately adjacent to the adenomatous lesions. The histological features of the experimental tumors were similar to those of human colorectal tumors. The similarity of adenocarcinomas and adenomatous lesions strongly suggests that the former might have originated from the latter.

- 3675 DOSE-RESPONSE AND ULTRASTRUCTURAL ALTERATIONS IN DIOXANE CARCINOGENESIS. INFLUENCE OF METHYLCHOLANTHRENE ON ACUTE TOXICITY. (E.) Argus, M. F. (U.S. Public Hlth. Service Hosp., New Orleans, La.), R. S. Sohal, G. M. Bryant, C. Hoch-Ligeti and J. C. Arcos. *Eur J Cancer* 9(4):237-243, 1973.

The hepatocarcinogenicity of dioxane in four groups of Sprague-Dawley rats (28-32 animals each) was a function of the total oral dose administered for 13 months. There were four incipient tumors in the 0.75% dioxane group, nine incipient tumors in the 1% dioxane group, 13 incipient tumors and three hepatomas in the 1.4% dioxane group, and 11 incipient tumors and 12 hepatomas in the 1.8% dioxane group. The progressive development of these tumors was studied histopathologically, and detailed electron microscopy was carried out on precancerous liver tissue and on hepatocellular carcinoma produced by dioxane. Precancerous changes, observed eight months following 1% dioxane, included fragmentation of the rough endoplasmic reticulum cisternae, detachment of the ribosomes and their dispersion in the cytoplasm; increase in the smooth endoplasmic reticulum, decrease in glycogen content of the hepatocytes, and an increase in lipid droplets within the hepatocytes. Acute toxicity experiments showed the LD₅₀ dose of dioxane in Sprague-Dawley rats is 5.60 g/kg, i.p. This is reduced to 5.18 g/kg by pretreatment with 3-methylcholanthrene (10 mg), suggesting that a metabolite is involved in

the toxicity and possibly carcinogenicity of dioxane. Both may be potentiated by enzyme inducers. Micro-iodimetric titrations indicated that the toxicity and carcinogenicity of dioxane cannot be imputed to a dioxane hydroperoxide.

- 3676 N-NITROSATION BY NITRITE ION IN NEUTRAL AND BASIC MEDIUM. (E.) Keefer, L. K. (Nat'l. Cancer Inst., Bethesda, Md.) and P. P. Roller. *Science* 181(4106):1245-1246, 1973.

Formaldehyde catalyzed the conversion of various secondary amines to nitrosamines in the pH range 6.4 to 11.0. Nitrosamine yields varied roughly according to steric accessibility of the nitrogen atom toward electrophilic attack. The order of decreasing reactivity was pyrrolidine \approx piperidine \approx dimethylamine $>$ diethylamine \approx di-*n*-propylamine \gg diisopropylamine. Chloral was also an effective catalyst. The reaction proceeds easily enough to have potential synthetic applications; the proposed mechanism involving initial interaction of aldehyde with the secondary amine could explain some reported anomalies regarding the synthesis of carcinogenic N-nitroso compounds *in vivo* and *in vitro*.

- 3677 THE PROTECTIVE EFFECT OF ESTRADIOL-17 β AGAINST POLYCYCLIC HYDROCARBON CYTOTOXICITY. (E.) Schwartz, A. G. (Albert Einstein Coll. Med., Bronx, N.Y.) *Cancer Res* 33(10):2431-2436, 1973.

7,12-dimethylbenz(a)anthracene (DMBA, 10^{-6} M) inhibited the growth of cultured epithelial rat liver cells and depressed thymidine- 3 H incorporation. In a series of methyl- and ethyl-substituted benz(a)anthracenes, there was significant correlation between the reported carcinogenicity of the derivative and its capacity to inhibit acid-insoluble thymidine- 3 H incorporation, ethyl-substituted derivatives being less inhibitory than the corresponding methyl-substituted compounds. Similar results were obtained with a diploid strain of rat lung fibroblasts, but a human cancer cell (HeLa) and a rat hepatoma (HTC) were insensitive to the cytotoxic effect of DMBA. In contrast to DMBA, a series of diazo dyes of varying hepatocarcinogenicities did not significantly inhibit thymidine- 3 H incorporation by liver epithelial cell cultures. Estradiol-17 β at 10^{-6} M protected liver and breast epithelial cultures from the inhibitory effect of DMBA on thymidine- 3 H incorporation. The other steroids tested (progesterone, testosterone, hydrocortisone, and deoxycorticosterone) had little or no protective effect at 4×10^{-5} M. The possible relationship of this protective effect of estradiol-17 β to clinical and epidemiological data in women is discussed.

- 3678 ON THE FUNCTIONAL STATE OF RAT LIVER RIBOSOMES FOLLOWING ADMINISTRATION OF THE CARCINOGEN DIMETHYLNITROSAMINE. (E.) Delpino, A. (Inst. Regina Elena Cancer Res., Rome, Italy) and U. Ferrini. *Eur J Cancer* 9(4):245-251, 1973.

Polyribosomes isolated from livers of male Wistar rats given a single dose of dimethylnitrosamine (100 mg/kg,

i.p.) were centrifuged in 15-40% linear sucrose gradients prepared in a low salt buffer and a high salt buffer. The depolymerization of the polyribosomes following a single dose of the carcinogen gave rise to a large amount of monoribosomes of the monomer type, as indicated by their susceptibility to be dissociated into 40S and 60S subunits by high ionic strength salt solutions. Tests on the capacity of these subunits to recombine into 80S couples and to support the polyuridylic-primed polyphenylalanine synthesis indicated that a fraction of these subunits is inactive. The ribosomes constituting the residual polysomes were unaffected by the drug. The correlation between these effects and the alkylating properties of the dimethylnitrosamine metabolites is briefly discussed.

- 3679 ACTIVITY OF RETICULOENDOTHELIAL SYSTEM IN METHYLCHOLANTHRENE-INDUCED AND TRANSPLANTED TUMOUR-BEARING RATS. (E.) Encut, I. (Oncol. Inst. Bucharest, Rumania), F. Liciu, L. Cioloca and E. Olaianos. *Neoplasma* 20(4):369-374, 1973.

The activity of the reticuloendothelial system (RES) to 20-methylcholanthrene (MCA)-induced and transplanted tumors in rats was assayed by the use of radioactive colloidal gold (^{198}Au). The evaluation of phagocytic function was given by determination of the percentage of injected radioactivity present in liver and spleen of the animals killed 15 minutes after the i.v. injection. Colloidal uptake/whole liver was significantly increased in inbred R III rats bearing primary 20-MCA-induced tumors, decreased in R III rats with isografted 2-MCA tumors, and highest in Wistar rats which had rejected tumor allografts. The uptake of colloidal radiogold in the spleen of rats with primary tumors was very close to the control values, while in rats with isografted tumors and in the animals which had rejected tumor allografts it was depressed. Splenic and hepatic enlargement occurred in primary tumor-bearing rats, was not observed as a rule in rats with isografted tumors, and did not occur in allografted rats. The significance of these modifications is discussed with regard to tumor growth and immune response of the host. The splenomegaly and hepatomegaly may be a manifestation of an immunologic reaction evoked by antigen(s) released from the tumors.

- 3680 CELLULAR REACTIONS OF O^6 -METHYLGUANINE, A PRODUCT OF SOME ALKYLATING CARCINOGENS. (E.) Miller, C. T. (Royal Cancer Hosp., London, England), P. D. Lawley and S. A. Shah. *Biochem J* 36(2):387-393, 1973.

Cultures of a purine-requiring mutant of Chinese hamster ovary cells (CHO-104b), randomly bred hamster embryo cells, or *Escherichia coli* B $_{S-1}$ were treated with nontoxic doses of ^3H -labelled O^6 -methylguanine. DNA and RNA were isolated and subjected to enzymic digestion to nucleosides at pH 8. The products of digestion were analysed by ion-exchange chromatography on Dowex 50 (NH_4^+ form) columns at pH 8.9. No ^3H -labelled O^6 -methylguanosine was detected in nucleic acid digests. ^3H -labelled O^6 -methylguanine was *O*-demethylated yielding [^3H]guanine in CHO-104b cells. Radioactivity in nucleic acid digests was associated

with thymidine, guanosine, deoxyguanosine, and an unidentified early eluting product. Reports of similar unidentified products from nucleic acids labelled with various agents are discussed. It is suggested that these products derive from incorporation of label from the methyl group of O^6 -methylguanine, or, less likely, from the degradation of the purine moiety thereof.

- 3681 INHIBITORY EFFECT OF CROSS-IMMUNITY ON AUTOTRANSPLANTATION OF METHYLCHOLANTHRENE-INDUCED RAT SARCOMAS. (E.) Usubuchi, I. (Hiroaki U. Sch. Med. Japan), H. Kudo, Y. Sobajima, T. Sato, Y. Kakisaka and S. Nishimura. *Tohoku J Exp Med* 110: 155-160, 1973.

The autologous transplantation of methylcholanthrene (MCA)-induced sarcomas in non-inbred rats, which had been immunized intensively by i.p. inoculation with various allogeneic tumors, was inhibited in 33 out of 65 cases tested. The difference of tissues from which allogeneic tumors originated did not influence the inhibitory effect on autotransplantation. In 10 out of 69 non-immunized controls, the autotransplantation of MCA-induced sarcomas proved to be negative. In addition, remarkable lymphoid cell infiltration was demonstrated in most of tumors induced by MCA in non-inbred rats which were immunized intensively with allogeneic tumors. The autologous transplantation was more markedly inhibited in the tumor infiltrated with massive lymphoid cells than in that not infiltrated.

- 3682 LUNG CARCINOGENESIS WITH 1-ACETYL-2-ISONICOTINOYLHYDRAZINE, THE MAJOR METABOLITE OF ISONIAZID. (E.) Toth, B. (U. Nebraska Med. Ctr., Omaha) and H. Shimizu. *Eur J Cancer* 9(4):285-289, 1973.

The continuous administration of 0.4% 1-acetyl-2-isonicotinoylhydrazine (AINH) in drinking water to 4 wk old randomly bred Swiss mice for the remainder of their lifetime gave rise to an increased incidence of lung tumors. Of 50 AINH-treated females, 37 animals developed 157 lung tumors with an incidence of 77% compared with 12% for controls. Of 50 AINH-treated males, 20 mice developed 108 lung tumors with an incidence of 58% compared with 10% for controls. The average daily intake of AINH was 21.6 mg for a female and 27.6 mg for a male. Ten of the 37 females with lung tumors had 58 adenomas and 16 adenocarcinomas and 27 mice had 83 adenomas. Six males had 29 adenomas and eight adenocarcinomas and 23 mice had 71 adenomas. Treatment with AINH had no apparent effect on the development of other types of tumors. The present study records for the first time the carcinogenicity of AINH, which in man is the major metabolite of the tuberculostatic drug isoniazid.

- 3683 ESTROGEN AND PROGESTOGEN BINDERS IN HUMAN AND RAT MAMMARY CARCINOMA. (E.) Terenius, L. (Dept. Med. Pharmacology, Uppsala, Sweden). *Eur J Cancer* 9(4):291-294, 1973.

Estrogen and progesterone binders (so-called receptors)

were measured in the high speed ultracentrifugal supernatant (cytosol) from 23 samples of human mammary carcinoma. Estrogen receptors were also measured in slices in 15 of these tumors. With one notable exception, the cytosol and slice techniques gave the same results. It was found that estrogen or progesterone cytosol receptors occurred in 12 tumors. In 6 cases there were substantial levels of both receptors, in 4 cases only estrogen receptors and in 1 case only progesterone receptors. Generally, there was no evidence of a correlation between the amounts of estrogen and progesterone receptors. Thus, the simultaneous measurement of both receptors in human breast carcinoma may give more information on the hormonal sensitivity of a tumor than measurements of only estrogen receptors which has been done previously. Breast tumors in rats induced by i.v. administration of 7,12-dimethylbenzanthracene contained cytosol receptors both for estrogens and progesterones.

- 3684 INDUCTION OF UNSCHEDULED DNA SYNTHESIS BY THE CARCINOGEN 7-BROMOMETHYLBENZ(A)ANTHRACENE AND ITS REMOVAL FROM THE DNA OF NORMAL AND XERODERMA PIGMENTOSUM LYMPHOCYTES. (E.) Slor, H. (Tel Aviv U. Med. Sch., Israel). *Mutat Res* 19(2):231-235, 1973.

The carcinogen 7-bromomethylbenz(a)anthracene (BBA), which can bind strongly to DNA, induces unscheduled DNA synthesis (DNA repair) in normal lymphocytes but almost none in lymphocytes from patients with Xeroderma pigmentosum (XP), an inherited disease known to be defective in excision repair of ultraviolet-damaged DNA. The ability of BBA to bind to DNA of normal lymphocytes and to lymphocytes from patients with Xeroderma pigmentosum (XP), its influence on unscheduled DNA synthesis, and its removal from the DNA of both cell types was studied. It was found that 20-30% of the BBA is bound to macromolecules other than DNA and that its binding to DNA is essentially complete after 30 min. The induction of unscheduled DNA synthesis by the carcinogen in XP lymphocytes was approximately 10% of that induced in normal lymphocytes. While 15-20% of the BBA was removed from the DNA of normal cells 6 hr after treatment, only 1-2% was removed from the DNA of XP cells. Thus, XP cells not only are defective in repairing UV-damaged DNA and excising thymine dimers but also fail to repair DNA damaged by certain carcinogens, and, most importantly, fail to remove the DNA-bound carcinogen, BBA. The malignancies observed in XP might be the result of UV irradiation, exposure to carcinogens that act like UV, or both. Restriction of most malignancies to sun-exposed areas of the skin implies that UV is the major etiological agent.

- 3685 INCREASED SENSITIVITY TO TUMOR INDUCTION WITH 3-HYDROXYXANTHINE BY MEANS OF IMMUNODEPRESSION. (E.) Teller, M. N. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.) and I. Smullyan. *Proc Am Assoc Cancer Res* 13(March): 73, 1972.

- 3686 ENZYMATIC N-ACETYLATION OF CARCINOGENIC ARYLAMINES BY SPECIES DISPLAYING DIFFERING ORGAN SUSCEPTIBILITIES. (E.) Lower, G. M., Jr. (Wisconsin Med. Sch., Madison) and G. T. Bryan. *Proc Am Assoc Cancer Res* 14(March):11, 1973.
- 3687 DETECTION OF CARCINOGENIC ACTIVITY IN DIFFERENT EXTRACTS OF BRACKEN FERN. (E.) Wang, C. Y. (U. Wisconsin Med. Sch., Madison), A. M. Pamukcu and G. T. Bryan. *Proc Am Assoc Cancer Res* 14(March):12, 1973.
- 3688 THE PROPERTIES OF O⁶-METHYLGUANINE IN TEM-PLATES FOR RNA POLYMERASE. (E.) Gerchman, L. L. (U. Maryland, Sch. Med., Baltimore) and D. B. Ludlum. *Proc Am Assoc Cancer Res* 14(March):13, 1973.
- 3689 RIBONUCLEASE ACTIVITY AND NEOPLASTIC TRANSFORMATION IN RAT LIVER DURING DIETHYLNITROSAMINE CARCINOGENESIS. (E.) Fontaniere, B. (Montreal Cancer Inst., Canada) and R. Daoust. *Proc Am Assoc Cancer Res* 14(March):17, 1973.
- 3690 ANALYSIS OF PRIMARY HEPATOMAS AND INITIAL TRANSPLANT GENERATIONS. (E.) Becker, F. F. (New York U. Sch. Med., N.Y.), K. M. Klein, S. R. Wolman, R. Asofsky and S. Sell. *Proc Am Assoc Cancer Res* 14(March):18, 1973.
- 3691 MEGALOCYTE INDUCTION IN LIVERS OF RATS TREATED WITH METHYLAZOXYMETHANOL ACETATE. (E.) Zedeck, M. S. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.), S. S. Sternberg and J. McGowan. *Proc Am Assoc Cancer Res* 14(March):7, 1973.
- 3692 ALTERED TRYPTOPHAN PYRROLASE (TP) AND TRYPTOPHAN METABOLISM IN RATS FED THE BLADDER CARCINOGEN N-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]-FORMAMIDE (FANFT). (E.) Leklem, J. E. (Wisconsin Med. Sch., Madison), R. A. Arend, S. M. Cohen and R. R. Brown. *Proc Am Assoc Cancer Res* 14(March):48, 1973.
- 3693 3'-ME-DAB-INDUCED INHIBITION OF POLYURIDYLIC ACID-DIRECTED POLYPHENYLALANINE SYNTHESIS BY FREE RIBOSOMES FROM RAT LIVER. (E.) Kizer, D. E. (Noble Fdn., Inc., Ardmore, Okla.) and J. A. Clouse. *Proc Am Assoc Cancer Res* 14(March):48, 1973.
- 3694 STUDIES ON INTERACTIONS BETWEEN POLYNUCLEOTIDES AND BENZO(a)PYRENE (BaP) IN THE PRESENCE OF ARYL HYDROCARBON HYDROXYLASE (AHH). (E.) Bogdan, D. P. (Dept. Biochem., Pharm., State U. New York, Buffalo) and Z. F. Chmielewicz. *Proc Am Assoc Cancer Res* 14(March):49, 1973.
- 3695 INHIBITION BY URETHAN OF FREE AND MEMBRANE-BOUND RIBOSOMAL RNA IN REGENERATING LIVER. (E.) Glazer, R. I. (Emory U., Atlanta, Ga.). *Proc Am Assoc Cancer Res* 14(March):53, 1973.
- 3696 INHIBITION OF CHEMICAL CARCINOGENESIS BY BUTYLATED HYDROXYANISOLE (BHA) AND THIURAM DISULFIDE DERIVATIVES. (E.) Wattenberg, L. W. (Dept. Path., U. Minnesota, Minneapolis). *Proc Am Assoc Cancer Res* 14(March):7, 1973.
- 3697 MALIGNANT TRANSFORMATION *IN VITRO* OF MOUSE FIBROBLASTS BY 7,12-DIMETHYLBENZ(A)ANTHRACENE, 7-HYDROXYMETHYLBENZ(A)ANTHRACENE AND BY THEIR "K-REGION" DERIVATIVES. (E.) Marquardt, H. (Sloan-Kettering Inst., New York, N.Y.), J. E. Sodergren, P. Sims and P. L. Grover. *Proc Am Assoc Cancer Res* 14(March):7, 1973.
- 3698 ANTIOXIDANTS DECREASE CARCINOGEN INDUCED CHROMOSOME BREAKAGE. (E.) Shamberger, R. J. (Cleveland Clin., Ohio). *Proc Am Assoc Cancer Res* 14(March):9, 1973.
- 3699 INDUCTION OF TUMORS IN MONKEYS BY CHEMICAL CARCINOGENS - CORRELATION OF SERUM ALPHAFETOPROTEIN AND APPEARANCE OF LIVER TUMORS. (E.) Adamson, R. H. (Natl. Cancer Inst., Bethesda, Md.), P. Correa, C. F. Smith, S. T. Yancey and D. W. Dalgard. *Proc Am Assoc Cancer Res* 14(March):42, 1973.
- 3700 INHIBITION OF SULFHYDRYL-DEPENDENT ENZYMES WITH PHENANTHRENE EPOXIDE. (E.) Hutcheson, E. T. (U. Tennessee Med. Units, Memphis) and J. L. Wood. *Proc Am Assoc Cancer Res* 14(March):65, 1973.
- 3701 EFFECTS OF p-HYDROXYPROPIOPHENONE (PHP) ON BINDING AND METABOLISM OF p-DIMETHYLAMINO-AZOBENZENE (DAB) IN RAT LIVER. (E.) Gordon, E. B. (New York U. Med. Ctr., N.Y.), B. L. Van Duuren and A. Sivak. *Proc Am Assoc Cancer Res* 14(March):66, 1973.
- 3702 MAMMARY CARCINOGENESIS IN NEONATALLY ANDROGENIZED, AND OVARECTOMIZED RATS. (E.) Christakos, S. (Roswell Park Mem. Inst., Buffalo, N.Y.), D. Sinha and T. L. Dao. *Proc Am Assoc Cancer Res* 14(March):67, 1973.
- 3703 CELL-CYCLE DEPENDENCY OF CHEMICAL ONCOGENIC TRANSFORMATION IN CULTURE. (E.) Bertram, J. S. (McArdle Lab., Madison, Wisc.) and C. Heidelberger. *Proc Am Assoc Cancer Res* 14(March):70, 1973.
- 3704 DNA REPAIR AFTER TREATMENT OF MOUSE SKIN CELL CULTURES WITH β -PROPIOLACTONE (BPL). (E.) Hennings, H. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 14(March):70, 1973.
- 3705 MUTAGENICITY OF DERIVATIVES OF THE ONCOGEN 3-HYDROXYXANTHINE. (E.) McCuen, R. W. (Sloan-Kettering Inst., New York, N.Y.), G. Stöhrer, F. M. Sirotnak and G. B. Brown. *Proc Am Assoc Cancer Res* 14(March):74, 1973.

- 3706 A BIOLOGIC MODEL FOR LARGE BOWEL CARCINOGENESIS. (E.) Reuber, M. D. (U. Maryland, Baltimore) and T. A. Hill. *Proc Am Assoc Cancer Res* 14(March):55, 1973.
- 3707 ISOLATION AND BIOLOGICAL ACTIVITY OF AN L-METHIONINE DEGRADING ENZYME, L-METHIONINE- α -DESAMINO- γ -MERCAPTOMETHANE-LYASE (L-METHIONINASE). (E.) Kreis, W. (Sloan-Kettering Inst., New York, N.Y.) and C. Hession. *Proc Am Assoc Cancer Res* 14(March):56, 1973.
- 3708 BINDING OF ^3H -BENZO(A)PYRENE TO DNA FROM HAMSTER TRACHEAL EPITHELIAL CELLS. (E.) Genta, V. M. (Natl. Cancer Inst., Bethesda, Md.), D. G. Kaufman, M. B. Sporn and U. Saffiotti. *Proc Am Assoc Cancer Res* 14(March):57, 1973.
- 3709 CILIA IN THE EPITHELIUM OF THE URINARY BLADDER DURING EXPERIMENTAL CARCINOGENESIS. (E.) Yalciner, S. (St. Vincent Hosp., Worcester, Mass.) and G. H. Friedell. *Proc Am Assoc Cancer Res* 14(March):58, 1973.
- 3710 LACK OF EVIDENCE FOR FORMATION OF DIAZOMETHANE FROM 5-(3,3-DIMETHYL-1-TRIAZENO)-IMIDAZOLE-4-CARBOXAMIDE. (E.) Shiota, F. N. (VA Hosp., Minneapolis, Minn.), H. T. Nagasawa and N. S. Mizuno. *Proc Am Assoc Cancer Res* 14(March):58, 1973.
- 3711 EFFECT OF VITAMIN A ON INDUCTION AND GROWTH OF SQUAMOUS CELL TUMORS IN THE RESPIRATORY TRACT. (E.) Nettesheim, P. (Oak Ridge Natl. Lab., Tenn.), M. V. Cone and M. L. Williams. *Proc Am Assoc Cancer Res* 14(March):59, 1973.
- 3712 CARCINOGENESIS BY DIAZOACETIC ESTER. (E.) Stenback, F. G. (Eppler Inst. Res. Cancer, Omaha, Neb.), H. Garcia and B. T. So. *Proc Am Assoc Cancer Res* 14(March):62, 1973.
- 3713 EFFECT OF DIETARY FAT ON RAT MAMMARY DEVELOPMENT. (E.) Chan, P.-C. (American Hlth. Fdn., New York, N.Y.). *Proc Am Assoc Cancer Res* 14(March):64, 1973.
- 3714 FREE AND BOUND RIBOSOMES IN METHYLCHOLANTHRENE INDUCED LIVER GROWTH. (E.) Argyris, T. S. (Upstate Med. Ctr., Syracuse, N.Y.) and R. Heinemann. *Proc Am Assoc Cancer Res* 14(March):6, 1973.
- 3715 ENHANCEMENT OF PULMONARY CARCINOGENESIS IN SNELL'S MICE AND ITS ABSENCE IN C57 BLACK MICE AFTER CHRONIC INHALATION OF CIGARETTE SMOKE. (E.) Leuchtenberger, C. (Swiss Inst. Exp. Cancer Res., Lausanne), R. Leuchtenberger and J. Rossier. *Proc Am Assoc Cancer Res* 14(March):6, 1973.
- 3716 TRANSFORMATION OF STRAIN-2 GUINEA PIG CELLS IN CULTURE BY CHEMICAL CARCINOGENS. (E.) Evans, C. H. (Natl. Cancer Inst., Bethesda, Md.) R. L. Nelson and J. A. DiPaolo. *Proc Am Assoc Cancer Res* 14(March):76, 1973.
- 3717 PROXIMATE CARCINOGEN INDUCED DNA REPAIR IN HUMAN DIPLOID FIBROBLASTS. (E.) Lieberman, M. W. (Natl. Cancer Inst., Bethesda, Md.), M. C. Poirier and C. C. Harris. *Proc Am Assoc Cancer Res* 14(March):77, 1973.
- 3718 MECHANISMS OF THE INHIBITION OF THE CARCINOGENICITY OF *N*-2-FLUORENYLACETAMIDE OR *N*-HYDROXY-2-FLUORENYLACETAMIDE BY *p*-HYDROXYACETANILIDE. (E.) Grantham, P. H. (Natl. Cancer Inst., Bethesda, Md.), L. C. Mohan, E. K. Weisburger and J. H. Weisburger. *Proc Am Assoc Cancer Res* 14(March):79, 1973.
- 3719 CARCINOGENICITY OF CLINICALLY USED ANTICANCER AGENTS. (E.) Prejean, J. D. (Mem. Inst. Pathol., Birmingham, Ala.), D. P. Griswold, J. C. Peckham, A. E. Casey, E. K. Weisburger and J. H. Weisburger. *Proc Am Assoc Cancer Res* 14(March):79, 1973.
- 3720 MAMMARY CARCINOGENESIS BY TOPICAL APPLICATIONS OF FLUORENYLHYDROXAMIC ACIDS. (E.) Malejka-Giganti, D. (VA Hosp., Minneapolis, Minn.) and H. R. Gutmann. *Proc Am Assoc Cancer Res* 14(March):81, 1973.
- 3721 TRANSPLACENTAL CARCINOGENESIS IN HAMSTERS. (E.) Rustia, M. (U. Nebraska Med. Ctr. Omaha) and P. Shubik. *Proc Am Assoc Cancer Res* 14(March):81, 1973.
- 3722 THE COCARCINOGENIC ACTIVITY OF NON-CARCINOGENIC AROMATIC HYDROCARBONS. (E.) Goldschmidt, B. M. (New York U. Med. Ctr., N.Y.), C. Katz and B. L. Van Duuren. *Proc Am Assoc Cancer Res* 14(March):84, 1973.
- 3723 INTERACTION OF HYDROXYLATED BENZOPYRENES WITH GENES. (E.) Yamamoto, N. (Temple U. Sch. Med., Philadelphia, Pa.), Y. Tagashira and M. Ueda. *Proc Am Assoc Cancer Res* 14(March):85, 1973.
- 3724 FACILITATION OF TUMOR GROWTH BY INTRA-TUMOR INJECTION OF BCG & *VIBRIO CHOLERAE* NEURAMINIDASE (VCN). (E.) Sparks, F. C. (VA Hosp., Sepulveda, Ca.) and J. H. Breeding. *Proc Am Assoc Cancer Res* 14(March):86, 1973.
- 3725 CARCINOGENESIS WITH 1-ACETYL-2-ISONICOTINOYLHYDRAZINE, THE MAIN METABOLITE OF ISONICOTINIC ACID HYDRAZIDE. (E.) Toth, B. (U. Nebraska Coll. Med., Omaha) and H. Shimizu. *Proc Am Assoc Cancer Res* 14(March):92, 1973.

- 3726 THE CARCINOGENICITY OF SODIUM CYCLAMATE IN COMBINATION WITH OTHER ONCOGENIC AGENTS. (E.) Rudali, G. (Curie Radium Inst. Fdn., Paris, France), I. Muranyi-Kovacs, E. Coezv and L. Aussepe. *Proc Am Assoc Cancer Res* 14(March):93, 1973.
- 3727 ARA-C PRODUCED TRANSFORMATION IN HAMSTER FETAL CELLS. (E.) Benedict, W. F. (Children's Hosp., Los Angeles, Calif.) and R. Kouri. *Proc Am Assoc Cancer Res* 14(March):94, 1973.
- 3728 APPARENT INHIBITION OF BLADDER CARCINOGENESIS IN THE RAT BY ALLOPURINOL. (E.) Romas, N. (Inst. Cancer Res., Columbia U., New York, N.Y.), B. Fingerhut, P. Feigelson and R. Veenema. *Proc Am Assoc Cancer Res* 14(March):95, 1973.
- 3729 MEMBRANE EFFECTS OF PHORBOL ESTERS. (E.) Wenner, C. (Roswell Park Mem. Inst., Buffalo, N.Y.), H. Kimelberg, E. Mayhew, D. Jacobs and J. Mackney. *Proc Am Assoc Cancer Res* 14(March):96, 1973.
- 3730 TYPE C VIRUS FROM CELL CULTURES OF CHEMICALLY INDUCED HEPATOMAS. (E.) Gebert, R. (Columbia U., New York, N.Y.) and I. B. Weinstein. *Proc Am Assoc Cancer Res* 14(March):96, 1973.
- 3731 *IN VITRO* TRANSFORMATION: COMBINED ACTION OF CHEMICALS AND RADIATION. (E.) DiPaolo, J. A. (Nat'l. Cancer Inst., Bethesda, Md.), B. C. Casto, P. J. Donovan, N. C. Popescu and C. Goodheart. *Proc Am Assoc Cancer Res* 14(March):96, 1973.
- 3732 STUDIES ON THE METABOLISM AND CARCINOGENICITY OF BENZO[a]PYRENE IN THE SKIN OF VARIOUS STRAINS OF MICE. (E.) Somogyi, A. (Dept. Biochem., Drug Metabolism, Hoffmann-LaRoche Inc., Nutley, N.J.), S. Banerjee, M. M. Jacobson, J. Spranger, L. Achor, R. Kuntzman and A. H. Conney. *Proc Am Assoc Cancer Res* 14(March):111, 1973.
- 3733 CONJUGATES OF THE N-HYDROXY METABOLITES OF AROMATIC AMINES IN DOG URINE. (E.) Radomski, J. L. (U. Miami, Sch. Med., Fla.), A. A. Rey and R. Brill. *Proc Am Assoc Cancer Res* 14 (March):126, 1973.
- 3734 BIOSYNTHESIS OF NITROSAMINES: REACTION OF SODIUM NITRITE WITH DIMETHYLGLYCINE PRO- DUCES NITROSOSARCOSINE. (E.) Friedman, M. A. (Hlth. Sci. Division, Virginia Commonwealth U., Richmond) and H. McClanahan. *Proc Am Assoc Cancer Res* 14 (March):127, 1973.
- 3735 SPECIFIC INHIBITION OF RIBOSOMAL RNA SYNTHESIS OF CELL CULTURES BY DAUNORUBICIN. (E.) Diez, J. (Children's Cancer Res. Fdn., Boston, Mass.) and T. Liu. *Proc Am Assoc Cancer Res* (March):128, 1973.
- 3736 AN ELECTRON MICROSCOPIC STUDY OF TYPE C VIRUS IN CELL CULTURES ESTABLISHED FROM CHEMICALLY INDUCED RAT HEPATOMAS. (E.) Orenstein, J. M. (Inst. Cancer Res., Columbia U., New York, N.Y.). *Proc Am Assoc Cancer Res* 14(March):99, 1973.
- 3737 EFFECT OF A NEW SERIES OF PROLACTIN INHIBITORS ON DMBA-INDUCED MAMMARY CARCINOMAS. (E.) Sweeny, M. J. (Lilly Res. Lab., Indianapolis, Ind.), J. A. Clemens, E. C. Kornfeld and G. A. Poore. *Proc Am Assoc Cancer Res* 14(March):101, 1973.
- 3738 LYMPHOCYTE TRANSFORMATION, PARADOXICAL EFFECT OF PHA STAPH EXTRACT, CON-A, PWM ON DNA SYNTHESIS. (E.) Nitschke, R. (Children's Hosp., Los Angeles, Calif.). *Proc Am Assoc Cancer Res* 14(March):101, 1973.
- 3739 EFFECT OF SODIUM ASCORBATE ON LUNG ADENOMA INDUCTION BY AMINES PLUS NITRITE. (E.) Mirvish, S. S. (U. Nebraska Med. Ctr., Omaha), A. Cardesa, L. Wallcave and P. Shubik. *Proc Am Assoc Cancer Res* 14(March):102, 1973.
- 3740 SUBCELLULAR COMPARTMENTATION OF GLUCOSE-6-PHOSPHATE DEHYDROGENASE AND ACID RIBONUCLEASE IN GROWING AND REGRESSING MAMMARY TUMORS. (E.) Cho-Chung, Y. S. (Nat'l. Inst. Hlth., Bethesda, Md.), B. Berghoffer and P. M. Gullino. *Proc Am Assoc Cancer Res* 14(March):102, 1973.
- 3741 THE EFFECT OF PORTACAVAL SHUNT ON EXPERIMENTAL MAMMARY CARCINOMA. (E.) Reichle, F. A. (Temple U. Hlth. Sci. Ctr., Philadelphia, Pa.), S. Alloy, M. Gruenstein and G. P. Rosemond. *Proc Am Assoc Cancer Res* 14(March):103, 1973.
- 3742 THE INFLUENCE OF 1,10-PHENANTHROLINE (1,10-PHE) ON ACUTE EFFECT OF ETHIONINE ON RAT LIVER. (E.) Brada, Z. (Papanicolaou Cancer Res. Inst. Miami, Fla.), M. S. Chen, O. E. Matos, S. Bulba and J. Cohen. *Proc Am Assoc Cancer Res* 14(March):106, 1973.
- 3743 ESTRIOLE INHIBITION OF PROCARBAZINE-INDUCED RAT MAMMARY CANCER. (E.) Lemon, H. M. (U. Nebraska Coll. Med., Omaha). *Proc Am Assoc Cancer Res* 14(March):117, 1973.
- 3744 THE LARGE WILD EUROPEAN HAMSTER AS A LUNG CANCER MODEL. (E.) Althoff, J. (U. Nebraska Med. Ctr., Omaha), U. Mohr and N. Page. *Proc Am Assoc Cancer Res* 14(March):122, 1973.
- 3745 CHARGE LOCALIZATION IN THE CARBONIUM ION OF BENZOPYRENES AND METHYLBENZANTHRACENES COUPLING WITH NUCLEOPHILIC COMPOUNDS. (E.) Cavalieri, E. (Eppley Inst. Res. Cancer, U. Nebraska, Omaha) and R. Auerbach. *Proc Am Assoc Cancer Res* 14(March):123, 1973.

- 3746 FREQUENCY OF TUMORS IN FISH POPULATING A POLLUTED WATER SYSTEM. (E.) Brown, E. R. (Chicago Med. Sch., Ill.), L. Keith, J. B. G. Kwapinski, J. Hazdra and P. Beamer. *Proc Am Assoc Cancer Res* 14(March):1, 1973.
- 3747 QUARTZ CARCINOGENICITY AT THE RAT SUBCUT-ANEOUS SITE. (E.) Bryson, G. (Cottage Hosp. Res. Inst., Santa Barbara, Calif.) and F. Bischoff. *Proc Am Assoc Cancer Res* 14(March):1, 1973.
- 3748 SMOOTH BUT NOT SOLID-STATE SURFACES IN EVANS RATS. (E.) Bischoff, F. (Cottage Hosp. Res. Inst., Santa Barbara, Calif.) and G. Bryson. *Proc Am Assoc Cancer Res* 14(March):1, 1973.
- 3749 ADDUCTS OF 2-ACETAMIDOPHENANTHRENE WITH METHIONINE AND ADENOSINE. A ROLE FOR FREE RADICALS IN AROMATIC AMINE CARCINOGENESIS. (E.) Scribner, J. D. (Pacific Northwest Res. Fdn., Seattle, Wash.) and N. K. Naimy. *Proc Am Assoc Cancer Res* 14(March):2, 1973.
- 3750 ENHANCEMENT OF B16 MELANOMA METASTASES BY 5-BROMO-2-DEOXYURIDINE(BuDR). (E.) Zeidman, I. (Sch. Med., U. Pennsylvania, Philadelphia). *Proc Am Assoc Cancer Res* 14(March):4, 1973.
- 3751 MEASUREMENT OF *IN VIVO* DAMAGE AND REPAIR OF RAT LIVER DNA INDUCED BY CHEMICAL CARCINOGENS. (E.) Michael, R. O. (Temple U. Sch. Med., Philadelphia, Pa.) and D. S. R. Sarma. *Proc Am Assoc Cancer Res* 14(March):5, 1973.
- 3752 INTERACTION OF CYCLIC NITROSAMINES WITH RAT LIVER DNA *IN VIVO*. (E.) Stewart, B. W. (Temple U. Sch. Med., Philadelphia, Pa.) and E. Farber. *Proc Am Assoc Cancer Res* 14(March):5, 1973.
- 3753 EFFECT OF 3-METHYLCHOLANTHRENE (3MC) ON GLUCURONIDE SYNTHESIS IN PERFUSED RAT LIVER. (E.) Hamada, N. (Roswell Park Mem. Inst., Buffalo, N.Y.) and T. Gessner. *Proc Am Assoc Cancer Res* 14(March):62, 1973.
- 3754 ENHANCED REGRESSION OF MAMMARY CANCERS IN RATS BY COMBINATION OF HIGH ESTROGEN OR OVARECTOMY AND ERGOCORININE. (E.) Quadri, S. K. (Dept. Physiology, Michigan St. U., East Lansing), G. S. Kledzik and J. Meites. *Proc Am Assoc Cancer Res* 14(March):18, 1973.
- 3755 EFFECT OF 7-BROMOMETHYLBENZ[a]ANTHRACENE AND 7-BROMOMETHYL-12-METHYLBENZ[a]ANTHRACENE ON PHYSICAL AND BIOLOGICAL PROPERTIES OF DNA AND EVIDENCE OF REPAIR OF LESIONS INDUCED. (E.) Maher, V. M. (Michigan Cancer Fdn., Detroit) and D. Douville. *Proc Am Assoc Cancer Res* 14(March):90, 1973.
- 3756 ELECTROPHILIC ACTIVITY OF CARCINOGENIC DI-ARYL ACETYLENIC CARBAMATES. (E.) Sharpe, I. D. (McArdle Lab. Cancer Res., Madison, Wisc.), J. A. Miller and E. C. Miller. *Proc Am Assoc Cancer Res* 14(March):19, 1973.
- 3757 FURTHER STUDIES ON THE METABOLISM AND CARCINOGENICITY OF SAFROLE. (E.) Wislocki, P. G. (McArdle Lab. Cancer Res., Madison, Wisc.), P. Borchert, E. C. Miller and J. A. Miller. *Proc Am Assoc Cancer Res* 14(March):19, 1973.
- 3758 SWITCHING FROM CIGARETTES TO SMALL CIGARS -- IS IT LIKELY TO REDUCE THE HEALTH HAZARDS OF SMOKING? (E.) Bross, I. (Roswell Park Mem. Inst., Buffalo, N.Y.) and J. Tidings. *Proc Am Assoc Cancer Res* 14(March):21, 1973.
- 3759 THE EFFECT OF 12-O-TETRADECANOYL-PHORBOL-13-ACETATE ON CYCLIC AMP LEVELS IN MOUSE SKIN. (E.) Belman, S. (New York U. Med. Ctr., N.Y.) and W. Troll. *Proc Am Assoc Cancer Res* 14(March):21, 1973.
- 3760 CHEMICAL INDUCTION OF LYMPHOMAS IN HAMSTERS. (E.) Izquierdo, J. N. (U. Rochester, N.Y.). *Proc Am Assoc Cancer Res* 14(March):23, 1973.
- 3761 INHIBITORY EFFECT OF MANGANESE UPON MUSCLE TUMORIGENESIS BY NICKEL SULFIDE. (E.) Sunderman, F. W., Jr. (U. Connecticut Sch. Med., Farmington), T. J. Lau and L. J. Cralley. *Proc Am Assoc Cancer Res* 14(March):24, 1973.
- 3762 STIMULATION OF THE SYNTHESIS OF MOUSE EPIDERMAL HISTONES BY TUMOR-PROMOTING PHORBOL ESTERS. (E.) Raineri, R. (McArdle Lab. Cancer Res., U. Wisconsin, Madison). *Proc Am Assoc Cancer Res* 14(March):111, 1973.
- 3763 TEST OF THE PROTEIN DELETION HYPOTHESIS OF CHEMICAL CARCINOGENESIS. (E.) Sani, B. P. (Inst. Cancer Res., Philadelphia, Pa.), D. M. Mott and S. Sorof. *Proc Am Assoc Cancer Res* 14(March):114, 1973.
- 3764 INCREASE OF HEPATIC MITOCHONDRIAL POPULATION DURING FEEDING NONCARCINOGENIC AZO DYES: APPARENT ABSENCE OF *DE NOVO* BIOSYNTHESIS. (E.) Arcos, J. C. (Tulane Med. Sch., New Orleans, La.), B. C. Wu and M. F. Argus. *Proc Am Assoc Cancer Res* 14(March):45, 1973.
- 3765 CHEMOTHERAPEUTIC, CARCINOGENIC AND CELL REGULATORY EFFECTS OF 1-PHENYL-3-MONO-METHYLTRIAZENE (PMT). (E.) Schmid, F. A. (Sloan-Kettering Inst., New York, N.Y.) and D. J. Hutchison. *Proc Am Assoc Cancer Res* 14(March):75, 1973.

- 3766 THE CONJUGATION OF 1,2-EPOXY-1,2,3,4-TETRAHYDRONAPHTHALENE WITH A SPECIFIC PROTEIN FRACTION OF HUMAN ENDOMETRIUM. (E.) Morrison, J. C. (U. Tennessee Med. Units, Memphis), W. D. Whybrew, C. M. Sobhy, W. C. Morrison, W. L. Wiser, S. A. Fish and E. T. Bucovaz. *Proc Am Assoc Cancer Res* 14(March):25, 1973.
- 3767 EFFECTS OF VITAMIN A COMPOUNDS AND ANALOGS ON MOUSE LIVER LYSOSOMES AND MIXED-FUNCTION OXIDASES. (E.) Hill, D. L. (Kettering-Meyer Lab., Southern Res. Inst., Birmingham, Ala.), S. Straight and T.-W. Shih. *Proc Am Assoc Cancer Res* 14(March):26, 1973.
- 3768 THE BINDING OF 7,12-DIMETHYLBENZ(a)ANTHRACENE (DMBA) TO REPLICATING AND NON-REPLICATING DNA *IN VIVO*. (E.) Bowden, G. T. (McArdle Lab. Cancer Res., Madison, Wisc.) and R. K. Boutwell. *Proc Am Assoc Cancer Res* 14(March):28, 1973.
- 3769 DAMAGE AND REPAIR OF HEPATIC DNA BY ETHYLATING CARCINOGENS. (E.) Cox, R. (Fels Res. Inst., Philadelphia, Pa.), I. Damjanov and C. C. Irving. *Proc Am Assoc Cancer Res* 14(March):28, 1973.
- 3770 ALTERED NUCLEAR-CYTOPLASMIC RNA TRANSPORT AND MALIGNANT TRANSFORMATION. (E.) Shearer, R. W. (Pacific Northwest Res. Fdn., Seattle, Wash.). *Proc Am Assoc Cancer Res* 14(March):29, 1973.
- 3771 THE ROLE OF THE K-REGION EPOXIDE OF 7-METHYLBENZ(a)ANTHRACENE IN THE *IN VIVO* BINDING OF THE PARENT HYDROCARBON. (E.) Brookes, P. (Ches-ter Beatty Res. Inst., London, England) and W. M. Baird. *Proc Am Assoc Cancer Res* 14(March):30, 1973.
- 3772 *IN VITRO* INHIBITION OF HISTONE METHYLATION. (E.) Baxter, C. S. (Coll. Med., U. Florida, Gainesville) and P. Byvoet. *Proc Am Assoc Cancer Res* 14(March):98, 1973.
- 3773 EFFECTS OF TRICHLOROPROPENE OXIDE ON ARYL HYDROCARBON HYDROXYLASE (AHH). (E.) Yang, C. S. (New Jersey Med. Sch., Newark). *Proc Am Assoc Cancer Res* 14(March):108, 1973.
- 3774 EFFECT OF CASEIN ON CARCINOGENESIS OF 3-METHYLCHOLANTHRENE IN MICE. (E.) Akamatsu, Y. (Med. Coll. Georgia, Augusta). *Proc Am Assoc Cancer Res* 14(March):108, 1973.
- 3775 DEVELOPMENT OF LUNG NEOPLASMS IN RATS TREATED WITH 7,12 DIMETHYLBENZ(a)ANTHRACENE. (E.) Blair, W. H. (Mercy Hosp., Chicago, Ill.), N. Otero and H. Rao. *Proc Am Assoc Cancer Res* 14(March):125, 1973.
- 3776 CARCINOGENICITY OF NICKEL BY DIFFERENT ROUTES. (E.) Furst, A. (Inst. Chem. Biol., U. San Francisco, Calif.) and D. Cassetta. *Proc Am Assoc Cancer Res* 14(March):31, 1973.
- 3777 *IN VITRO* ALKYLATION OF MOUSE SKIN CHROMATIN BY β -PROPIOLACTONE- ^{14}C . (E.) Segal, A. (New York U. Med. Ctr., N.Y.), M. Schroeder and B. L. Van Duuren. *Proc Am Assoc Cancer Res* 14(March):33, 1973.
- 3778 GASTRIC TUMORS IN MICE FED REFINED CORN OIL PLUS FREE FATTY ACIDS. (E.) Szepsenwol, J. (U. Puerto Rico Sch. Med., San Juan). *Proc Am Assoc Cancer Res* 14(March):35, 1973.
- 3779 *IN VIVO* ALKYLATION OF DNA BY CARCINOGENIC β - AND γ -LACTONES. (E.) Paul, J. S. (U. Texas, Southwestern Med. Sch., Dallas), J. P. Hieble, R. Roe and P. O'B. Montgomery. *Proc Am Assoc Cancer Res* 14(March):38, 1973.
- 3780 AN UNUSUAL MALIGNANT TUMOR FROM CELLS TRANSFORMED *IN VITRO* BY DIEPOXYBUTANE. (E.) Wolman, S. R. (New York U. Sch. Med., N.Y.), A. Sivak and M. LaRocca. *Proc Am Assoc Cancer Res* 14(March):38, 1973.
- 3781 COMPARATIVE EFFECTS OF AGE, STRAIN AND SPECIES ON THE ACTIVATION OF N-HYDROXY-2-FLUORENYLACETAMIDE (N-HYDROXY-FAA) BY SOLUBLE ACYL-TRANSFERASES. (E.) King, C. M. (Michael Reese Hosp., Chicago, Ill.) and C. W. Olive. *Proc Am Assoc Cancer Res* 14(March):41, 1973.

See also:

- * (Rev): 3604, 3611, 3613, 3619, 3620, 3632
- * (Viral): 3810, 3849, 3896
- * (Immun): 3930, 3950, 3992
- * (Path): 4054, 4057
- * (Epid-Biom): 4061, 4062, 4064

- 3782 ELECTRON MICROSCOPIC STUDY OF LYMPHATIC TISSUES OF MICE INOCULATED WITH A LEUKEMOGENIC EXTRACT FROM RADIATION-INDUCED TUMORS. (E.) Ricciardi-Castagnoli, P. (Ctr. Study Nuclear Energy, Mol, Belgium), J. M. Jadin and J. R. Maisin. *Cancer Res* 33(10):2476-2488, 1973.

Macroscopic, histological, and ultrastructural changes in lymphatic tissues, particularly that of the spleen, were studied in 4-wk-old male C57BL mice inoculated i.p. with a cell-free extract of lymphatic tissues from mice in which leukemia was originally radiation induced. Two groups of 200 mice each were used to study the survival time of leukemic and control mice. Other groups of mice were used to investigate the morphology of various tissues and to observe weight changes of the spleen over a 5- to 85-day period. Splenomegaly was observed macroscopically, followed by hypertrophy of lymph nodes, but no change of the thymus was apparent throughout the course of this infection. The histological appearance of the spleen showed hyperplasia of the germinal centers within the lymphatic nodules. The lymphoid cells were replaced progressively by proliferation of the reticular cells and to a lesser extent, by cells of the plasmocytic line. Typical type C particles were found in the splenic white pulp. During the early stages of this disease, the particles were concentrated in the extracellular spaces within the germinal centers. The infected cells were predominantly immunoblasts, plasmoblasts, plasmocytes, and to a lesser extent lymphoblasts, lymphocytes, and reticular cells. Viral particles could not be detected within megakaryocytes or erythrocytic precursor cells. A few intracisternal type A particles were observed in both plasmocytes and reticular cells.

- 3783 EFFECTS OF IONIZING RADIATION ON RNA METABOLISM IN CULTURED MAMMALIAN CELLS. I. SPECIFIC EFFECTS DURING DIVISION DELAY FOLLOWING EXPOSURE TO X IRRADIATION. (E.) Enger, M. D. (Cellular Molecular Radiobiol Group, Los Alamos Sci. Lab., N. M.), E. W. Campbell and R. A. Walters. *Biochim Biophys Acta* 324(1):120-132, 1973.

RNA metabolism was studied in cultured Chinese hamster cells during the 9 hr division delay period following 800 rads of X irradiation. The ability of RNA species to support translation at a normal rate was unaffected. During the delay period, an amount of stable RNA equal to that of the control population was synthesized. This RNA is functional as judged by increased polysome mass and maintenance of translation rate. In consequence, the division delayed population has a significantly greater content of protein and RNA per cell by the time division recurs. At 7 hr post-irradiation, incorporation of uridine into heterogeneous nuclear RNA is about 26% greater in the irradiated population. There is not a corresponding increase of incorporation into informosomal messenger-like RNA. However, there is a 25% increase in incorporation into mRNA associated with polysomes in a 7 hr post-irradiation population. Thus, an increased incorporation into heterogeneous nuclear RNA subsequent to X irradiation is accompanied by

an increase in incorporation into cytoplasmic poly-some-associated RNA but not by an increased incorporation into informosomal RNA.

- 3784 INDUCTION OF LEUKEMIA BY ^{131}I TREATMENT OF THYROID CARCINOMA. (E.) Brincker, H. (Radium Ctr., Odense, Denmark), H. S. Hansen and A. P. Andersen. *Br J Cancer* 28(3):232-237, 1973.

The medical records of 194 Danish patients with thyroid carcinoma treated with ^{131}I therapy (50-1313 mCi) from 1948 to 1972 were reviewed with respect to the subsequent development of leukemia. Two cases of myeloid leukemia were found, an incidence significantly ($P < 0.05$) higher than that expected based on published incidence rates. Combination of these cases with those previously reported in four other studies produced a total of 10 cases in 487 patients treated with ^{131}I . This represented a leukemia frequency of about 2% and indicated that a certain risk is associated with ^{131}I treatment of thyroid carcinoma.

- 3785 THE CYTOGENETICS OF URANIUM MINERS EXPOSED TO ^{222}Rn . (E.) Brandom, W. F. (St. Mary's Hosp., Grand Junction, Colo.), G. Saccomanno, P. C. Archer and V. E. Archer. *Mutat Res* 21(4):212, 1973.

- 3786 THE EFFECT OF LUNG IRRADIATION ON THE INCIDENCE OF PULMONARY METASTASES IN MICE. (E.) Brown, J. M. (Stanford U. Med. Ctr., Calif.). *Br J Radiol* 46(548):613-618, 1973.

- 3787 HEPATOSPLENIC TUMOR CAUSED BY THOROTRAST (STUDY OF GAMMA RADIATION). (Fr.) Duriez, R. (Dept. Atomic Hyg., C.R.S.S.A., Clamart, France), R. Delahaye, H. Frossard, G. Prat, P.-J. Metjes, P. Poutrain, J. Langlois, J.-P. Bernard and M. Faure. *Ann Med Interne (Paris)* 124(8-9):581-585, 1973.

- 3788 RADIATION CHEMISTRY OF NUCLEIC ACIDS: IDENTIFICATION OF THE MAJOR HYDROPEROXY THYMINES. (E.) Hahn, B. S. (Sch. Hygiene, Publ. Hlth., Johns Hopkins U., Baltimore, Md.) and S. Y. Wang. *Biochem Biophys Res Commun* 54(3):1224-1232, 1973.

- 3789 CANCER INDUCTION IN MAN FROM INTERNAL RADIOACTIVITY. (E.) Mays, C. W. (Radiology Division, U. Utah, Salt Lake City). *Health Phys* 25(6):585-592, 1973.

- 3790 TUMORIGENESIS BY MILLIPORE FILTERS IN MICE: HISTOLOGY AND ULTRASTRUCTURE OF TISSUE REACTIONS AS RELATED TO PORE SIZE. (E.) Karp, R. D. (Med. Sch., U. Minnesota, Minneapolis), K. H. Johnson, L. C. Buoen, H. K. G. Ghobrial, I. Brand and K. G. Brand. *J Natl Cancer Inst* 51(4):1275-1285, 1973.

- 3791 HEMATOPOIETIC DAMAGE CAUSED BY THORIUM RADIATION THERAPY. (Ger.) Stieglitz, R. (Dept. Hematol., Humboldt U., Berlin, Germany), M. Thiele, H. Stobbe and G. Wegener. *Folia Haematol* 100(1/2):95-103, 1973.
- 3792 EFFECTS OF CORTICOSTEROID AND RADIATION ON LYMPHOID TISSUE IN MICE. COMPARISON: AND MUTUAL INTERACTIONS. (E.) Lundin, P. (Inst. Path., U. Goteborg, Sweden) and B. Järplid. *Lymphology* 6(3):158-166, 1973.
- 3793 DISTRIBUTION OF ^{226}Ra IN THE BONES OF MICE. (E.) Kofranek, V. (Inst. Hygiene, Epidemiology, Prague, Czechoslovakia), O. Parizek, J. Machek, J. Thomas and J. Hanzlik. *Acta Radiol [Ther]* (Stockh) 12(4):353-367, 1973.
- 3794 TOTAL BODY IRRADIATION AND HUMAN CHROMOSOMES. III. CYTOGENETIC STUDIES IN PATIENTS WITH MALIGNANT HEMATOLOGIC DISEASES TREATED WITH TOTAL-BODY IRRADIATION. (E.) Goh, K.-O. (Med. Division, Oak Ridge Associated U., Tenn.). *Am J Med Sci* 266(3):179-186, 1973.
- 3795 A RADIATION-INDUCED BREAST CANCER. (E.) Warren, S. (New England Deaconess Hosp., Boston, Mass.). *Cancer* 32(4):991-993, 1973.
- 3796 DEVELOPMENT AND ELIMINATION OF PIGMENTED MOLES, AND THE ANATOMICAL DISTRIBUTION OF PRIMARY MALIGNANT MELANOMA. (E.) Nicholls, E. M. (U. New South Wales, Sydney, Australia). *Cancer* 32(1):191-195, 1973.
- 3797 CHARACTERIZATION OF A NEW LYMPHOID CELL TYPE DETECTED IN THE IRRADIATED MOUSE. (E.) Haot, J. (Anatomy Path. Lab., U. Liege, Belgium), E. H. Betz and L. Revesz. *Nature [New Biol]* 244(137):211-212, 1973.
- 3798 ALTERATIONS OF THYROID MORPHOLOGY IN MICE BY RADIOACTIVE PHOSPHORUS - EFFECT OF LARGE DOSE. (E.) Dev, P. K. (Radiation Biol. Lab., U. Rajasthan, Jaipur, India) and P. N. Srivastava. *Strahlentherapie* 145(6):674-679, 1973.
- 3799 ESTIMATION OF PREVALENCE RATES OF RADIOGENIC LEUKEMIAS IN RFM/U MICE. (E.) Elashoff, R. M. (Res. Systems Div., U. California, San Francisco), F. C. Ludwig and P. Hemphill. *Proc Soc Exp Biol Med* 143(4):1150-1152, 1973.
- 3800 LATE EFFECTS OF IRRADIATION ON THE THYROID GLAND IN MICE. III. COMPARISON BETWEEN IRRADIATION OF FOETUSES AND ADULTS. (E.) Walinder, G. (Div. Radiobiol., Res. Inst. Natl. Defence, Sundbyberg, Sweden) and A.-M. Sjöden. *Acta Radiol (Stockh)* 12(3):201-208, 1973.
- 3801 LEUKEMIA IN IRRADIATED PARABIOTIC RATS. (E.) Warren, S. (New England Deaconess hosp., Boston, Mass.), K. B. Udupa and R. N. Chute. *Proc Am Assoc Cancer Res* 14(March):8, 1973.
- 3802 UV PHOTOCARCINOGENESIS: MODIFICATION BY ANTI-LYMPHOCYTIC SERUM OR 6-MERCAPTOPYRINE. (E.) Nathanson, R. B. (Temple U. Hlth. Sci. Ctr., Philadelphia, Pa.), P. D. Forbes and F. Urbach. *Proc Am Assoc Cancer Res* 14(March):46, 1973.
- 3803 TUMOR INDUCTION IN THE SYRIAN HAMSTER BY FRACTIONATED X-IRRADIATION. (E.) Stenback, W. A. (Baylor Coll. Med., Houston, Tex.), M. E. Bryan and J. J. Trentin. *Proc Am Assoc Cancer Res* 14(March):85, 1973.
- 3804 THE EFFECT OF DOSE FRACTIONATION ON THE RECOVERY OF RADIATION-INDUCED ONCOGENESIS IN RAT SKIN. (E.) Albert, R. E. (New York U. Med. Ctr., N.Y.) and F. J. Burns. *Proc Am Assoc Cancer Res* 14(March):86, 1973.
- 3805 THE ONCOGENIC RESPONSE OF ANAGEN PHASE RAT SKIN TO ELECTRON RADIATION OF VARIOUS PENETRATIONS. (E.) Burns, F. J. (New York U. Med. Ctr., N.Y.), I. P. Sinclair and R. E. Albert. *Proc Am Assoc Cancer Res* 14(March):88, 1973.
- 3806 EFFECT OF IRRADIATION ON MOUSE SALIVARY GLANDS DURING THE PREREPLICATIVE PHASE OF ISOPROTERENOL-STIMULATED DNA SYNTHESIS. (E.) Sasaki, T. (Tokyo Med. Dental U., Japan) and M. Toda. *Cancer Res* 32(12):2807-2812, 1972.

See also:

- * (Rev): 3608, 3640
 * (Chem): 3652, 3731

- 3807 A SMALL INTRANUCLEAR VIRUS RECOVERED FROM LYMPHATIC TISSUE OF A BOVINE WITH MALIGNANT LYMPHOMA. (E.) Burroughs, A. L. (Department Infectious Dis. Physiological Sci., Kansas State U., Manhattan) and G. H. Cardinet. *Arch Gesamte Virusforsch* 42(1):67-77, 1973.

Madin-Darby Bovine Kidney (MDBK) cells were permanently infected with a virus following inoculation and long-term incubation with lymphatic tissue from a 7-yr-old bovine with lymphocytic type of malignant lymphoma. The virus, passed to a calf by dripping a suspension into its eyes, nostrils, and mouth, was recovered from lymphatic tissue 106 days later. One of six calves inoculated with a quantity of concentrated virus suspension developed persistent lymphocytosis in approximately two yr. Permanently infected MDBK cells of a line infected for several months were agglutinated by both concanavalin A and wheat germ lipase contaminant. As with some RNA tumor viruses, there was no cytopathic effect but rather a slow multiplication with apparent maturation occurring at a cell membrane, accompanied by a proliferation of cells in the infected culture in greater numbers than in the non-infected control cultures. Electron micrographs of thin sections of cells revealed many virions in the nuclei. Enveloped particles ranged from 47 to 58 mμ in diameter. Autoradiographic studies resulted in labelling the intranuclear virions of infected cells with uridine-5-³H. Incorporation of isotope was not inhibited by FUDR and actinomycin D. Until nucleic acid can be obtained from purified preparations from chemical identification, it can be concluded only inferentially from these results that the viral core should be ribonucleic acid.

- 3808 MORPHOLOGIC OBSERVATIONS OF BRAIN TUMORS IN PD4 HAMSTERS INDUCED BY FOUR STRAINS OF AVIAN SARCOMA VIRUS. (E.) Burger, P. C. (Duke U. Med. Ctr., Durham, N.C.), D. D. Bigner and D. J. Self. *Acta Neuropathol (Berl)* 26(1):1-21, 1973.

Brain tumors were induced in inbred PD4 hamsters with each of four strains of Avian Sarcoma Virus: CT-559, Bratislava-77 (B-77), Schmidt-Ruppin (SR), and Bryan. Based on light and electron microscopic observations the neoplastic tissues found in these tumors were identified as: (1) glial, (2) a distinctive large cell, and (3) sarcomatous. The glial neoplasms consisted almost entirely of masses of astrocytes in a subependymal position. The large cell tissue was a striking feature in some of the tumors. Although the cell of origin for this tissue could not be positively identified, certain features suggested that these cells were derived from glia. The cerebral sarcomas occurred as small nodules near the pial surface or occasionally as large intracerebral masses. The CT-559 strain produced lesions with appreciably greater numbers of large-and-small cells, while the SR strain produced lesions with relatively few. This difference, however, was difficult to quantitate precisely by the methods used. Replicating virus was not seen in any of the tumors. The viral genome was present in the tumors, however, since it was rescued from four of the B-77-induced tumors by co-cultivation with chicken cells.

- 3809 TUMOUR REGRESSION AND RELAPSE IN MICE INOCULATED WITH MURINE SARCOMA VIRUS (MOLONEY). (E.) Perk, K. (Nat'l. Cancer Inst., Bethesda, Md.), E. Russel, K. L. Smith and J. B. Moloney. *Lab Anim* 7(3):255-263, 1973.

The oncogenic effect of murine sarcoma virus (Moloney) was tested in 648 mice of 7 different strains and three different age groups: newborn, and 4- and 6-wk old. Local sarcomas were induced in all mice, although strain, age or dose dependent variations were found. Four forms of development occurred: progressive lethal; lethal but long persistent; complete tumor regression; tumor recurrence after regression. Virus activity was highest in the progressively-growing and in the recurrent tumors, while in the long-persistent tumors or in tissue at the site of regressed tumors, little or no virus could be detected.

- 3810 ACTIVATION OF THE RAUSCHER LEUKEMIA VIRUS BY METALS. (E.) Gainer, J. H. (Nat'l. Inst. Environ. Hlth. Sci., Research Triangle Park, N.C.). *J Nat'l Cancer Inst* 51(2):609-613, 1973.

Cobaltous sulfate (0.01 M) and lead acetate (0.03 M) (but not cupric chloride, nickel sulfate, or the organic arsenical roxarsone), administered in drinking water to CD-1 male mice, induced splenomegaly after injection of Rauscher leukemia virus (300 plaque-forming U/mouse, i.p.). Enlarged spleens contained high titers of virus. Sodium arsenite, cadmium chloride, ferric chloride, and particularly mercuric chloride were suspected inducers of splenomegaly. At least one spleen in each of the first three groups was enlarged (550 mg or more), while spleens of mercuric chloride-treated mice were almost significantly larger than those of controls. Roxarsone treatment resulted in a slight decrease in splenic size. Untreated virus control mice resisted infection in that they did not develop splenomegaly, and virus was not recovered from their spleens.

- 3811 AVIAN TUMOR VIRUS RNA: A COMPARISON OF THREE SARCOMA VIRUSES AND THEIR TRANSFORMATION-DEFECTIVE DERIVATIVES BY OLIGONUCLEOTIDE FINGERPRINTING AND DNA-RNA HYBRIDIZATION. (E.) Lai, M. M. C. (Dept. Molecular Biol. Virus Lab., U. California, Berkeley), P. H. Duesberg, J. Horst and P. K. Vogt. *Proc Nat'l Acad Sci USA* 70(8):2266-2270, 1973.

Several sarcoma viruses contain two classes of 30-40S subunits of their 60-70S RNA. A larger one, referred to as class *a* subunits, appears to contain the genetic information required for transformation of fibroblasts; a smaller one, called class *b* subunits, is characteristic of transformation-defective (td) derivatives of sarcoma viruses as well as of the leukosis viruses investigated. To determine the chemical basis for the difference between class *a* and *b* subunits, the RNAs of three nondefective avian sarcoma viruses, B77 and Prague and Schmidt-Ruppin strains of Rous sarcoma virus, were compared with those of their td derivatives, td B77, td PR-C and td SR-A. Oligonucleotide fingerprinting showed that 1) all (20-25) large T1 RNase-resistant oligonucleo-

tides present in class *b* subunits of transformation-defective viruses have homologous counterparts in the class *a* subunits of corresponding nondefective sarcoma viruses and that 2) class *a* subunits contain a few (one or two) additional oligonucleotides that are not present in class *b*. Conversely, the oligonucleotide fingerprints of avian tumor viruses of different strains and subgroups are very different. Cross hybridization of classes *a* and *b* RNA of sarcoma virus B77 with DNA transcribed from a corresponding transformation-defective virus, td B77, demonstrated that the two RNAs share at least 60% and differ by about 10% of their sequences.

ference in rate or total amount of virus bound. Furthermore, studies with virions of vesicular stomatitis virus phenotypically mixed within an envelope containing Friend leukemia virus protein show no differences in penetration or replication of vesicular stomatitis virus. These results strongly suggest that host restriction of Friend leukemia virus is due to an intracellular event in the viral replication cycle and does not involve an interaction of the virus envelope and the cell surface.

3814 PROPERTIES OF MOUSE LEUKEMIA VIRUSES. IV. HEMAGGLUTINATION ASSAY AND CHARACTERIZATION OF HEMAGGLUTININATING SURFACE COMPONENTS. (E.) Witter, R. (Max-Planck Inst. Virus Res., Tübingen, Germany), H. Frank, V. Moennig, G. Hunsmann, J. Lange and W. Schäfer. *Virology* 54(2):330-345, 1973.

A hemagglutination (HA) assay for mouse leukemia virus (MuLV) concentrates was developed. Before assay, the virus was pre-treated with neuraminidase and phospholipase C. The assay gave linear dose-response data between concentrations of approximately 10^{11} and 8×10^{12} particles per ml. One HA unit was equivalent to approximately 5×10^7 virus particles in those preparations where surface projections (knobs) were readily detectable on the virions. When knobs were removed during purification or by bromelain treatment, the HA activity was reduced or eliminated. Bromelain-treated virus also lacked glycoprotein component(s), did not absorb neutralizing MuLV antibody, and was non-infectious. Thus, the HA substructure is clearly a surface component of the virion and has an apparent association with the surface knobs, glycoprotein(s), and type-specific antigens. The treatment with neuraminidase and phospholipase C, as required for demonstration of HA activity, damaged the envelope of the virion but did not release hemagglutinating subunits or surface knobs. Active hemagglutinin recovered from enzyme-treated virus by degradation with Tween 80 and ether and density gradient centrifugation was stable under various physical treatments but was heat sensitive. Active hemagglutinin could also be recovered from non-enzyme-treated virus by similar procedures. Concentrated preparations of avian myeloblastosis virus and feline leukemia virus agglutinated chicken and sheep erythrocytes, respectively, but in both cases the specific HA activities were low and the reactions could not be inhibited by specific antisera.

3815 ONCOGENIC TRANSFORMATION OF HAMSTER EMBRYO CELLS AFTER EXPOSURE TO INACTIVATED HERPES SIMPLEX VIRUS TYPE 1. (E.) Duff, R. (Milton S. Hershey Med. Ctr., Hershey, Pa.) and F. Rapp. *J Virology* 12(2):209-217, 1973.

The *in vitro* transformation of hamster embryo fibroblasts by herpes simplex virus type 1 (HSV-1) after exposure of the virus to UV irradiation is described. Cell transformation was induced by 2 of 12 strains of HSV-1 tested for transforming potential. Cells transformed by the KOS strain of HSV-1 were not oncogenic when injected s.c. into newborn Syrian hamsters. However, cells transformed by HSV-1 strain 14-012 induced tumors in 18 of 38 (47%) of the newborn ham-

3812 REVERSIBLE SPLENOmegaly WITH LOW VIRUS PRODUCTION INDUCED IN HYPERIMMUNE MICE BY PARABIOSIS WITH FRIEND-VIRUS-INFECTED MICE. (E.) Takizawa, K. (Inst. Med. Sci., Tokyo, Japan), H. Yoshikura, H. Sugano and T. Yamamoto. *Int J Cancer* 12(2):502-510, 1973.

Marked splenomegaly was induced in hyperimmune DDD mice by 10-day parabiosis with Friend leukemia virus (FLV)-infected DDD mice. Cell migration was not the main cause of the splenomegaly since parabiosis of FLV-infected DDD mice with congenic DD-Fv^r mice did not induce splenomegaly in the latter. Infectious center assay showed that few of the proliferated spleen cells in the hyperimmune mice were virus producers. In mice with splenomegaly, no suppression of primary immune response to sheep erythrocytes was induced by FLV infection. Separation from the nonimmunized partners after 10-day parabiosis resulted in regression of the splenomegaly in the hyperimmune mice. However, the prolongation of parabiosis resulted in persistent splenomegaly accompanied by an increase in virus-producing cells, suppression of the primary immune response to sheep erythrocytes, and appearance of typical Friend cells in the spleen. It is suggested that FLV-infected mice produce a splenomegaly-inducing substance that is not neutralized immunologically in hyperimmune mice. Curtailment of parabiosis within 10 days cuts off the transfer of the substance from the partner, while prolonged parabiosis results in the establishment of the productive mechanism of the substance even in hyperimmune mice.

3813 HOST RESTRICTION OF FRIEND LEUKEMIA VIRUS. ROLE OF THE VIRAL OUTER COAT. (E.) Krontiris, T. (Albert Einstein Coll. Med., Bronx, N.Y.), R. Soeiro and B. N. Fields. *Proc Natl Acad Sci USA* 70(9):2549-2553, 1973.

Host restriction of oncogenesis of RNA tumor viruses *in vivo* is associated with several gene loci. One of these genes, the *Fv-1* locus in mice, is expressed *in vitro* and may be studied in mouse-embryo cultures that are restrictive or permissive for replication of Friend leukemia virus. Two strains of Friend leukemia virus, N- or B-tropic, show reciprocal ability to replicate successfully in NIH Swiss (N-type) or BALB/c (B-type) cells that differ at the *Fv-1* locus. These two strains of virus and two cell lines form a system to measure host restriction *in vitro*. Measurement of adsorption of Friend leukemia virus to permissive or restrictive cells reveals no dif-

sters injected. Several of the tumors appeared pathologically similar to an adenocarcinoma; none was of the fibrosarcoma type previously reported for HSV-2 transformed hamster embryo cells. HSV-specific antigens were found in the cytoplasm of cells transformed by both virus strains. Sera from tumor-bearing hamsters contained HSV-1- and HSV-2-neutralizing antibodies as well as antibodies which reacted specifically with HSV antigens by the indirect immunofluorescence technique. Hamster oncornavirus antigens were not detected by immunofluorescence methods. These observations represent the first evidence of the oncogenic potential of HSV-1.

- 3816 SUPPRESSION OF ESTABLISHED FRIEND VIRUS LEUKEMIA BY STATOLON. VII. RELATIVE ROLES OF INTERFERON AND THE IMMUNE RESPONSE IN DEVELOPMENT OF FV-DORMANT INFECTIONS. (E.) Toy, S. T. (Dept. Microbiol., Thomas Jefferson U., Philadelphia, Pa.), O. S. Weislow and E. F. Wheelock. *Proc Soc Exp Biol Med* 143(3):726-732, 1973.

Interferon inducers, statolon, Newcastle disease virus, and polyinosinic-polycytidylic double-stranded RNA (poly I:poly C) were examined in female DBA/2J mice, 6-8 wk old, for their ability to clear virus from the blood of Friend virus (FV)-infected mice, to reestablish immunologic responsiveness and to suppress FV to a dormant state. Interferon was found to be operative for only a short period of time, accounting for clearance of FV from the blood. However, interferon induction and transient clearance of FV from the blood of leukemic mice are not sufficient by themselves to induce remissions. Of the interferon inducers tested, only statolon abrogates immunodepression, stimulates FV-cytotoxic antibody, and converts a rapidly lethal FV infection to a dormant state resulting in prolonged clinical remissions.

- 3817 FORMATION OF SYNCYTIA IN HUMAN LYMPHOBLASTOID CELLS INFECTED WITH TYPE C VIRUSES. (E.) Hampar, B. (Natl. Cancer Inst., Bethesda, Md.), K. H. Rand, R. A. Lerner, B. C. Del Villano, Jr., R. M. McAllister, L. M. Martos, J. G. Derge, C. W. Long and R. V. Gilden. *Virology* 65(2):453-463, 1973.

Infection of human lymphoblastoid cells with either of two feline type C viruses (FeLV and RD-114) resulted in the rapid appearance of syncytial cells attributable to cell fusion. Syncytia disappeared from infected cultures in approximately 7 days. By 3-5 wk after infection, a second cycle of syncytia occurred followed by the establishment of chronically infected cultures in the absence of further syncytial formation. The percentage of cells involved in the two cycles of syncytia was dependent on the input virus dose. With high input doses, the initial cycle of syncytia involved 15-20% of the cells, while the second cycle involved 5% or less of the cells. With low virus input doses, the percentage of syncytial cells were greatest during the second cycle. Cultures chronically infected with RD-114 virus were resistant to fusion induced by RD-114 virus or FeLV. Cultures chronically infected with FeLV were still susceptible to fusion induced by RD-114 virus but

not by FeLV. Treatment of RD-114 virus with B-propiolactone (BPL) destroyed the infectivity of the virus when tested in RD (nonlymphoid) cells, although the BPL-inactivated virus could still induce fusion of human lymphoblastoid cells. Surprisingly, lymphoblastoid cells infected with BPL-inactivated RD-114 virus showed a second cycle of syncytia at 3-5 wk after infection which was followed by the appearance of the infectious virus and the establishment of chronically infected cultures. A small percentage of syncytial cells induced by RD-114 virus showed synthesis of Epstein Barr (EB) virus antigens. In the case of nonproducer cells (Raji), the synthesis of EB virus-associated early antigen(s) suggested activation of the repressed virus genome following fusion induced by RD-114 virus. Disappearance of syncytia was accompanied by disappearance of EB virus associated antigens. Once established, Raji RD-114 carrier cultures showed no evidence of EB virus antigen synthesis.

- 3818 TRANSCRIPTION OF SIMIAN VIRUS 40. II. HYBRIDIZATION OF RNA EXTRACTED FROM DIFFERENT LINES OF TRANSFORMED CELLS TO THE SEPARATE STRANDS OF SIMIAN VIRUS 40 DNA. (E.) Ozanne, B. (Cold Spring Harbor Lab., N.Y.), P. A. Sharp and J. Sambrook. *J Virol* 12(1):90-98, 1973.

The amount of simian virus 40 (SV40) DNA present in various SV40-transformed mouse cell lines and "revertants" isolated from them was determined. The number of viral DNA copies in the different cell lines ranged from 1.35-8.75 copies/diploid quantity of host DNA and from 2.2-14 copies/cell. The revertants had the same number of viral DNA copies/diploid quantity of mouse cell DNA as their parental cell lines; however, they showed an increased number of viral DNA copies/cell due to their increased amount of DNA. By using separated strands of SV40 DNA, the extent of each DNA strand transcribed into stable RNA species was determined for the transformed and "revertant" cell lines. From 30-80% of the "early" strand and from 0-20% of the "late" strand was present as stable RNA species in the cell lines tested. There was no alteration in the pattern of the stable viral RNA species present in three concanavalin A-selected revertants, whereas in a fluorodeoxyuridine-selected revertant there was less viral-specific RNA present in the cells.

- 3819 VARIANTS OF SIMIAN VIRUS 40-TRANSFORMED 3T3 CELLS THAT ARE RESISTANT TO CONCANAVALIN A. (E.) Ozanne, B. (Cold Spring Harbor Lab., N.Y.). *J Virol* 12(1):79-89, 1973.

By treating populations of simian virus 40 (SV40)-transformed 3T3 cells with concanavalin A (Con A), variants have been isolated which are resistant to the killing action of the lectin. The variants resemble 3T3 cells morphologically and in some of their growth characteristics; are not agglutinated by high concentrations of Con A or wheat germ agglutinin, but can be rendered agglutinable by treatment with low concentrations of trypsin; bind the same

number of Con A molecules as 3T3 or SV3T3 cells; cannot be transformed by SV40 and are resistant to focus formation after infection with murine sarcoma virus; contain SV40-specific T antigen and RNA and; yield wild-type SV40 virus after heterokaryon formation with BS-C-1 cells.

- 3820 MALIGNANT LYMPHOMA IN COTTONTOP MARMOSETS AFTER INOCULATION WITH EPSTEIN-BARR VIRUS. (E.) Shope, T. (Yale U. Sch. Med., New Haven, Conn.), D. Dechario and G. Miller. *Proc Natl Acad Sci USA* 70(9):2487-2491, 1973.

Neoplasia resembling human malignant lymphoma, reticulum cell sarcoma type, occurred in cottontop marmosets inoculated (1 ml divided into 3 aliquots given i.v., i.p. and s.c.) with materials containing Epstein-Barr virus (EBV). One of the 4 animals that received autologous cells $1.2-3.0 \times 10^8$ cells/ml transformed *in vitro* by EBV developed lymphoma in mesenteric lymph nodes 7.5 months after inoculation. Three of 4 marmosets inoculated with cell-free EVB developed lymphoma. The latent period for detectable tumor formation after addition of virus was 31-46 days. Immunosuppressive drugs given with the virus accelerated the course of disease. Nevertheless, malignant lymphoma occurred in an animal given only cell-free virus. Six of 8 marmosets inoculated with the virus demonstrated antibodies to the virus. None of 4 marmosets not exposed to the virus, including two that received immunosuppressive drugs, developed tumors or antibodies to EBV. Virus antigen detectable by immunofluorescence was found in 5% of cells shed from one tumor maintained in organ culture. These results imply that EBV virus is capable of inducing malignant lymphoma in at least one primate species. Additional evidence is required before its oncogenic capacity in this host can be accepted without reservation.

- 3821 TRANSFORMATION OF SWISS 3T3 CELLS BY MURINE SARCOMA VIRUS IS FOLLOWED BY DECREASE IN A GLYCOLIPID GLYCOSYLTRANSFERASE. (E.) Mora, P. T. (Natl. Cancer Inst., Bethesda, Md.), P. H. Fishman, R. H. Bassin, R. O. Brady and V. W. McFarland. *Nature [New Biol]* 245(147):226-229, 1973.

Specific activities of the four glycolipid glycosyltransferases involved in ganglioside biosynthesis were assayed by *in vitro* radiometric techniques at various times following morphologic transformation of mouse 3T3 cell cultures by the Moloney isolate of murine sarcoma virus (MSV). A 50% decrease in N-acetylgalactosaminyl (galNAc) transferase specific activity occurred two to three days after transformation. Galactosyl (gal) transferase specific activity, however, increased about three-fold compared to uninfected controls. Concomitant thin-layer chromatographic analysis of gangliosides extracted from infected cells showed an 80% reduction in those gangliosides containing N-acetylgalactosamine. Infection with murine leukemia virus (MLV) alone produced no significant changes in galNAc or gal transferase specific activities. Studies using MSV-positive, MLV-negative (S^+L^-) cloned 3T3 cells

suggested that the decrease in ganglioside biosynthesis was related only to transformation by MSV. Enzyme assay of mixtures of homogenates from untransformed and MSV-transformed cells showed that decreased galNAc transferase activity was not due to increased hydrolase activity for GM₃ or UDP-galNAc in transformed cells. This observed decrease of galNAc transferase activity of RNA virus-transformed cells was similar to that previously observed for DNA virus-transformed established mouse cell lines.

- 3822 AUTOGENOUS IMMUNITY TO ENDOGENOUS RNA TUMOR VIRUS. RADIOIMMUNE PRECIPITATION ASSAY OF MOUSE SERUM ANTIBODY LEVELS. (E.) Ihle, J. N. (Carcinogenesis Program, Oak Ridge Natl. Lab., Tenn.), M. Yurconic and M. G. Hanna, Jr. *J Exp Med* 138(1):194-208, 1973.

The radioimmune precipitation assay using 3H -labeled AKR leukemia virus was applied to the detection and quantitation of natural serum antibodies directed against endogenous murine leukemia virus envelope antigens. B6C3F₁ and BALB/c mice, which have low natural incidences of leukemia and lymphoma, and AKR mice, which have a high incidence, were used in this study. Sera from mice of various age groups were assayed. A marked difference in age-associated levels of the autogenous immune response to endogenous murine leukemia virus was detected, and the quantitative differences among these strains were inversely related to the incidence of lymphoma. The radioimmune precipitation test as applied was 500 times more sensitive than virus neutralization. That the observed reactions are specific is suggested by several lines of evidence, including the non-reactivity of normal hamster and absorbed rat serum, the positive reaction of absorbed rat anti-AKR serum, the inhibition of precipitation of labeled virus by purified unlabeled virus, and isopycnic gradient analysis of the reactive products.

- 3823 MATERNAL VACCINATION WITH FORMALIN-INACTIVATED GROSS LYMPHOMA VIRUS IN RATS AND TRANSFER OF IMMUNITY TO OFFSPRING. (E.) Ioachim, H. L. (Lenox Hill Hosp., New York, N.Y.), M. L. Gimovsky and S. E. Keller. *Proc Soc Exp Biol Med* 144(2):376-379, 1973.

The ability of female rats to confer immunity on their offspring following three weekly i.p. inoculations of formalin-inactivated Gross lymphoma virus (GLV) before or during mating was studied. Newborn litters from vaccinated mothers and from nonvaccinated controls were challenged by live GLV injection within the first four days. Whereas 19 of 21 rats born to nonvaccinated controls developed lymphoma within 95 days after GLV injection, only 3 of 57 rats born to vaccinated mothers were diseased. There was no difference between the two groups in the mean latency period for tumor formation. Of the three schedules used to inactivate GLV, the best results were obtained with formalin 1:4000 at 37 C in the presence of 1 M MgCl₂. None of 15 offspring born to mothers so vaccinated had lymphoma. Sera from mothers vaccinated with either inactivated or live GLV showed *in vitro*

neutralizing activity against GLV. Vaccination of females with an inactivated oncogenic virus represents a model which may be applicable to the prevention of oncogenesis in other animal systems.

3824 ISOLATION OF LEUKEMIA VIRUS FROM A TRANSPLANTED LINE OF HUMAN CELL CULTURES. (Rus.)

Zhdanov, V. M. (D. I. Ivanovskii Inst. Virol., Moscow, USSR), V. D. Solov'ev, T. A. Bektemirov, F. P. Filatov and A. F. Bykovskii. *Vestn Akad Med Nauk SSSR* (4):3-5, 1973.

Since C-type virus particles were consistently found in cultures of transplantable human J-96 cells, which were originally obtained from leukocytes of a patient with leukemia, an attempt was made to isolate and characterize these particles. Cultures were grown for 3-4 days on medium No. 199 which was then replaced with the same medium containing mitomycin C (0.5 µg/ml) or 5-bromodeoxyuridine (100-200 µg/ml). After 24 hr this culture medium was replaced by medium No. 199. Culture fluid was decanted after 3-5 days, and the supernatant was centrifuged to remove cells and cell debris. The virus particles were then isolated by placing clear culture fluid in an ultracentrifuge at 30,000 rpm for 3-4 hr. Sediment obtained in this way was subjected to gradient density centrifugation in a 15-60% sucrose gradient at 25,000 rpm for 3 hr, and the fraction with a density of 1.16 g/ml was investigated. C-particles were detected under the electron microscope; some of the virions had disintegrated and strands of ribonucleoproteins were seen. Reverse transcriptase activity was demonstrated in this fraction; reaction products were identified as DNA and DNA-RNA hybrids. It is unlikely that the C-particles are identical with Rauscher leukemia virus, Bittner virus, or Rous sarcoma virus since no reactions were obtained on immunodiffusion with antisera for these viruses. Contamination with a bovine leukemia virus cannot be ruled out since bovine serum was added to the culture fluid, but the existence of this virus is only hypothetical. It is concluded that C-particles isolated from human J-96 cell cultures are probably human leukemia virus.

3825 THE HISTOGENESIS OF TUMOURS INDUCED IN GOLDEN HAMSTERS BY MURINE SARCOMA VIRUS-HARVEY (MSV-H). (E.) Hallows, R. C. (Imperial Cancer Res. Fund, London, England), F. C. Chesterman and D. G. West. *Int J Cancer* 12(3):705-721, 1973.

Cytological and ultrastructural studies were conducted on the pathological lesions induced in newborn golden hamsters following s.c. inoculation with a cell-free filtrate of cultured mouse 3T3 cells infected with murine sarcoma virus Harvey (MSV-H). MSV-H-induced lesions included: 1) cystic changes developing 2-3 wk post inoculation in lymph nodes adjacent to the inoculation site; 2) granulomatous nodules in the skin, s.c. tissue, lymph nodes, and muscle adjacent to the inoculation site; 3) mast cell tumors seen from 3-6 wk post-inoculation in the lymph nodes affected by

cystic changes; and 4) infiltrating, partly granulomatous mesenchymal tumors appearing 12-24 wk post inoculation in the paws and limb muscles distant from the inoculation site. The only sites in which virus-like particles were seen by electron microscopy were cytoplasmic vacuoles within the endothelial cells lining the cysts and cystic serous fluid which showed type H (radial) virus-like particles. The cystic fluid also contained monocytes, phagocytic histiocytes, polymorphs, and RBC. The predominant pleomorphic cell type seen by electron microscopy in the MSV-H induced lesions was morphologically related to the histiocyte.

3826 STUDY OF THE ETIOLOGY OF HUMAN LEUKEMIA IN BABOONS (PAPIO HAMADRYAS). (Rus.)

Iakovleva, L. A. (No affiliation), B. A. Lapin, V. N. Fomenko, M. T. Ivanov and V. F. Shchekolodkin. *Vestn Akad Med Nauk SSSR* (4):20-31, 1973.

Leukemia was induced in 4 of 6 infant baboons and 5 or 8 mothers by inoculating them with whole citrated blood or plasma from untreated patients with acute leukemia or chronic myeloid leukemia. Infants were inoculated *in utero* and mothers, during pregnancy. Both infants and mothers received a second i.m. injection shortly after delivery. Infants were given a total of 3-5 ml and mothers, 15-30 ml of blood or plasma. In each case the 2 injections were of blood or plasma from patients with the same form of leukemia. The leukemia which developed in these baboons was an aleukemic reticulosis characterized by reticulocyte and lymphoblast infiltration and spontaneous remissions. Although the WBC was very high (60,000-75,000) shortly before death, 60% of the cells were polymorphonuclear leukocytes with only a few metamyelocytes and myelocytes; monocytes increased to 13-19%. Leukemia was transmitted to 19 adolescent baboons and 2 brown macaques by i.p. inoculation of 5-8 ml blood from 3 baboons (2 adult females and 1 infant) with leukemia. Some of these animals apparently recovered spontaneously. By means of the indirect immunofluorescence test with antisera to WBC of leukemia patients, a new membrane antigen was detected in WBC in 7 adult females and some of the animals who developed leukemia after inoculation with blood from infected baboons. C-particles were isolated from plasma and detected in 48-hr blood cell cultures from one baboon with leukemia; Laidlow type mycoplasma was detected in cells from the spleen and lymph nodes. These findings indicate that the leukemia induced in baboons is viral in origin, but further immunological investigations are needed to rule out the possibility that the leukemia might be caused by a latent leukemia virus in the baboons.

3827 A COMPARISON OF DNA BINDING PROTEINS FROM NORMAL AND VIRUS-TRANSFORMED MOUSE CELLS. (E.) Rubio, V. (Flow Lab., Rockville, Md.), W.-P. Tsai, K. Rand and C. Long. *Int J Cancer* 12(3):545-550, 1973.

The DNA binding proteins present in normal and SV40- or murine sarcoma virus (MSV)-transformed

3T3/B and 3T6 mouse embryo fibroblast cells were analyzed using DNA cellulose chromatography and SDS polyacrylamide gel electrophoresis. Cellular proteins were prelabeled with either ^3H or ^{14}C -leucine. Exponentially growing normal mouse 3T6 cells possessed two major DNA binding proteins (P6 and P8) and two minor proteins (P3 and P5) as previously reported. Exponentially growing MSV-transformed 3T6 cells synthesized increased amounts of P3, P5, and P6 compared with normal 3T6 cells, while SV40-transformed 3T3 cells grown under identical conditions had increased levels of P3. Nonproducer 3T3 cells carrying a defective MSV genome and grown under "step-down" conditions of serum depletion produced normal levels of P5, P6, and P8 proteins. These same three proteins were present in decreased amounts in normal 3T3 cells grown under similar conditions.

3828 INTRACEREBRAL TRANSPLANTATION OF HUMAN RHABDOMYOSARCOMA CELLS INTO FETAL AND NEWBORN KITTENS. (E.) Gardner, M. B. (U. Southern California Sch. Med., Los Angeles), E. Y. Johnson, S. Rasheed and R. M. McAllister. *Int J Cancer* 12(3):563-567, 1973.

Uninfected or virus-infected human rhabdomyosarcoma cells (RD) were transplanted intracerebrally into fetal kittens at 45-55 days gestation or into newborn kittens less than 24 hr old, and attempts were made to recover an RD-114-like virus. A total of 13 tumors arising from inoculation of RD cells, RD-114 virus, or RD cells infected with feline leukemia virus (FeLV) or murine sarcoma virus (MSV) were harvested for tissue culture and virologic studies. None of six RD transplant tumors at different passage levels showed evidence of RD-114 virus, MSV, or FeLV infection when assayed by complement fixation tests, for RNA-directed DNA polymerase activity, or by electron microscopy for C-type particles. RD cells shedding RD-114, FeLV, or MSV before inoculation continued to shed the homologous virus upon growth as intracerebral transplant tumors in fetal or newborn kittens. Contamination or "pick-up" of RD-114 virus occurred only in two of three RD-114 cell transplant tumors. In addition to the six RD transplant tumors, no RD-114 virus was detectable in two RD-FeLV transplant tumors or in two RD-MSV transplant tumors.

3829 LOCALIZATION AND INDUCTION OF THE HUMAN THYMIDINE KINASE GENE BY ADENOVIRUS 12. (E.) McDougall, J. K. (Dept. Cancer Studies, U. Birmingham, England), R. Kucherlapati and F. H. Ruddle. *Nature [New Biol]* 245(145):172-175, 1973.

Experiments were performed on adenovirus type 12 infected hybrid cells from the WL24a-2-A line in order to use the virus-specified lesion to further identify the translocated segment of the human 17 chromosome and also to attempt the recovery of virus-induced deletions. Infection of the hybrid cells with either 1 or 100 plaque forming units (PFU) per cell of adenovirus 12 resulted in the induction of chromosome aberrations which were apparently random

in the mouse genome. Non-random, virus-induced gaps were found in the mouse/17 translocated chromosome present in the WL24a-2-A mouse/human hybrid cell line. This gap occurs in the proximal part of the long arm of this chromosome. It was also found that the human gene for thymidine kinase (TK) is closely associated with the site of the virus-induced gap and the breakage of the chromosome in the area of this gap region results in loss or retention of the gene. The fact that breaks occur in the uncoiled region at either side of the TK locus indicates that the visible virus-induced gap is larger than the TK gene. It is possible that genes for other virus-induced proteins may be located in this area or that transcription of the TK gene involves initiation and termination sites at some distance from the gene itself.

3830 THE EFFECT OF BLEOMYCIN ON SV40 DNA: CHARACTERISTICS OF BLEOMYCIN ACTION WHICH PRODUCES A SINGLE SCISSION IN A SUPERHELICAL FORM OF SV40 DNA. (E.) Umezawa, H. (Inst. Microbiol. Chem., Tokyo, Japan), H. Asakura, K. Oda, S. Hori and M. Hori. *J Antibiot (Tokyo)* 26(9):521-527, 1973.

Alkaline sucrose gradient centrifugation studies were conducted to determine the effect of bleomycin on the molecular integrity of ^3H -thymidine prelabeled SV40 DNA under various experimental conditions. The effects of bleomycin were dose-dependent, at concentrations under 2 $\mu\text{g}/\text{ml}$. Conversion of 53 S (form I) to 16-18S (form II) DNA by 2 to 4 $\mu\text{g}/\text{ml}$ bleomycin was optimal at 0 C, pH 9.1. Activity was less at 37 C and did not occur at pH values below 6 or above 13. Addition of unlabeled SV40, *E. coli* or *Pseudomonas* DNA to the reaction mixture reduced the effectiveness of bleomycin. Addition of *E. coli* tRNA, however, had no effect. 2-Mercaptoethanol (1 mM) stimulated by 20-fold and CuSO_4 (1 mM) almost abolished the effect of bleomycin. Unlike previous reports, 10 mM EDTA was without effect on bleomycin activity in this assay system.

3831 DNA METHYLATION IN NORMAL AND TUMOUR VIRUS-TRANSFORMED CELLS IN TISSUE CULTURE. I. THE LEVEL OF DNA METHYLATION IN BHK21 CELLS AND IN BHK21 CELLS TRANSFORMED BY POLYOMA VIRUS (PyY CELLS). (E.) Rubery, E. D. (Dept. Biochem., Cambridge U., England) and A. A. Newton. *Biochim Biophys Acta* 324(1):24-36, 1973.

The extent of methylation of DNA cytosine moieties to 5-methylcytosine was determined in BHK21 cells and was compared to that occurring in polyoma virus-transformed BHK21 (PyY) cells. Conversion of ^{14}C -labeled cytidine to DNA 5-methylcytosine, as determined by the amount of radioactivity in product isolated from DNA hydrolysates by proper chromatography, was twice as great in PyY cells as in BHK21 cells when calculated on the basis of total DNA cytosine. Results of experiments utilizing ^{14}C -thymidine indicated that 5-methylcytosine was not being produced from amination of DNA thymine residues. Further studies showed that DNA methylation occurred only during the period of DNA synthesis. The rate of methylation of DNA in cultures

synchronized with deoxyadenosine appeared to plateau as the rate of DNA synthesis became maximum.

- 3832 BURKITT'S TUMOR. (E.) Alexander, L. L. (No affiliation) and N. M. L. Atkins. *J Natl Med Assoc* 65(5):386-390, 1973.

Observations on Burkitt's tumor in African children are reported together with current tumor treatment techniques. Because adequate radiation facilities have not been available in Africa, extensive use is made of chemotherapeutic agents. Three Ghanaian children are presented who have had no recurrence of tumor following treatment with i.v. cytoxan or a cyclic sequential regimen of cytoxan, cyclophosphamide (40 mg/kg i.v. every 2-3 wk), vincristine (1.4 mg/m² i.v. on day 1), methotrexate (15 mg/m² p.o. on days 1-4), cytosine arabinoside (250 mg/m² daily for 3 days, and intrathecal methotrexate (10 mg/wk X 4). Burkitt's tumor in these children involved, resp., the left maxilla and orbital areas, abdomen and right orbit, and left mandible. Patients with Burkitt's syndrome can now hope for a normal life expectancy with radiotherapy and various chemotherapeutic agents used alone or in combination.

- 3833 ACTIVATION OF TYPE C RNA TUMOR VIRUS EXPRESSION IN TUMORS INDUCED BY CELL CULTURES TRANSFORMED BY POLYOMA VIRUS. (E.) Rhim, J. S. (Microbiological Associates, Inc., Bethesda, Md.) and R. J. Huebner. *Proc Soc Exp Biol Med* 144(1):210-214, 1973.

NIH Swiss mouse embryo cells transformed by polyoma virus were negative for virus and group-specific (gs) antigens of type C RNA tumor virus. When the transformed cells were transplanted into s.c. tissues of newborn NIH Swiss mice the tumors were positive for gs antigens but negative for infectious virus. Similarly, polyoma virus transformed hamster embryo cells led to hamster leukemia virus gs antigen after transplantation into newborn virus-free hamsters. Since mouse and hamster cells have been shown to contain the complete genome of the RNA tumor viruses, it is suggested that a DNA tumor (polyoma) virus transformation in cell cultures and expressed (or activated) gs antigen of type C RNA tumor virus in tumors may be related events.

- 3834 NEUTRALIZING ANTIBODIES TO EPSTEIN-BARR VIRUS IN HEALTHY POPULATIONS AND PATIENTS WITH INFECTIOUS MONONUCLEOSIS. (E.) Hewetson, J. F. (Children's Hosp. Philadelphia, Pa.), G. Rocchi, W. Henle and G. Henle. *J Infect Dis* 128(3):283-289, 1973.

Sera from healthy donors and individuals with primary Epstein-Barr virus (EBV) infections were titrated for EBV-neutralizing antibodies, and the results were compared with those obtained in indirect immunofluorescence assays for antibodies to EB-viral capsid antigens (anti-VCA) and to EBV-induced early antigens (anti-EA). Sera from more than 100 healthy donors without anti-VCA failed to show neutralizing

activity, whereas, with one exception, anti-VCA-positive sera from nearly 150 donors neutralized the virus. The ratios between the neutralizing and anti-VCA titers varied considerably, however, indicating that different antibodies were measured. The general correspondence with neutralizing activity explains why anti-VCA serves as a dependable indicator of immunity to infectious mononucleosis (IM). Accordingly, sera taken before infection from individuals who subsequently developed IM or seroconverted with respect to anti-VCA antibodies without evident or significant signs of disease were devoid of neutralizing activity. Titers of anti-VCA antibody usually attained peaks early in the course of the disease (within two wk) and subsequently declined to lower, persistent levels, whereas maximal neutralizing titers were reached in six to seven wk and showed no significant declines thereafter. Because of their slow development, diagnostically significant increases in titers of neutralizing antibody (\geq fourfold) were observed more frequently than corresponding increments in anti-VCA antibody.

- 3835 PREVENTION OF MURINE SARCOMA VIRUS ONCOGENESIS IN OFFSPRING OF IMMUNIZED FEMALE MICE. (E.) Chieco-Bianchi, L. (Inst. Path. Anat., U. Padova, Italy), D. Collavo, G. Biasi and A. Colombatti. *Br J Cancer* 238-244, 1973.

The protective effects of prenatal maternal immunization were studied in seven- to ten-day-old offspring BALB/c mice by challenge with graded doses of murine sarcoma virus (MSV)-induced tumor cell extracts. Forty-five percent of the mice born to and nursed by females immunized with two prenatal and one postnatal injection of MSV-induced tumor cell extract developed tumors following challenge as compared to 97% of the mice born to controls. The mean latent period for tumor induction, which was the same for both groups, was dose-dependent. Also, the incidence of spontaneously regressing tumors was significantly higher in mice born to immunized mothers. Females immunized six to 32 wk before mating could also confer protection to their offspring. Females immunized after parturition, however, could not. Only three of 14 MSV-injected litters whose mothers had been previously exposed to virus while nursing infected offspring showed a reduced tumor incidence following challenge. Sera from both immunized mothers and their suckling offspring showed the presence of MSV neutralizing antibody as determined by an *in vitro* focus reduction assay. When cell-free extracts from mice which developed leukemia after MSV inoculation were tested for oncogenicity in one-wk-old mice, four of the six extracts induced typical MSV tumors and the remaining two induced leukemias.

- 3836 TUMOUR-ASSOCIATED ANTIGEN IN THE SERUM OF RATS WITH LARGE ROUS SARCOMA VIRUS-INDUCED TUMOURS. (E.) Ridi, R. E. (Inst. Exp. Biol. Genetics, Czechoslovak Acad Sci., Prague), and J. Bubenik. *Folia Biol (Praha)* 19(4):273-280, 1973.

Cells of RSV-induced transplantable rat sarcoma,

RSL, were incubated *in vitro* with sera from rats bearing large progressively growing RSL sarcomas and inoculated into syngeneic recipients. Using the doses of 0.05-0.3 ml/rat, 13 out of 18 sera transferred resistance to tumor growth, three transferred enhancement and two sera were without detectable effect. In the membrane immunofluorescence tests, no antibody activity was detected in 14 out of 18 sera, one serum gave positive results and the remaining three sera were not tested. On the other hand, in all four out of the 18 sera, which were examined for the presence of tumor-associated antigen (TAA), the serum TAA was demonstrated by immunofluorescence after adsorption to viable syngeneic normal rat lymph node cells. It is suggested that the serum TAA is released from the tumor mass in a manner analogous to the carcino-embryonic antigens detectable in the serum of individuals with tumors of the gastrointestinal tract. The serum antigen can immunize, if injected together with tumor cells in the tumor-neutralization tests, and/or interfere with the development of active immunity depending on the dose, physical-chemical form, and the immunological status of the recipient.

- 3837 AN ONCOGENIC VIRUS CARRIED BY HAMSTER KIDNEY CELLS. (E.) Mayo, J. (Dept. Radiobiol., Natl. Atomic Energy Commission, Buenos Aires, Argentina), J. L. Lombardo, A. J. P. Klein-Szanto, C. J. Conti and J. L. Moreira. *Cancer Res* 33(10):2273-2277, 1973.

A filterable oncogenic factor was isolated from ascitic tumors induced in 12- to 20-week old adult X-irradiated hamsters by inoculation of BHK 21 clone 13 cells. This agent reproduces a tumor in hamsters 22 days after inoculation. Its oncogenic characteristics are preserved even after the ascitic tumor cells have been subcultured *in vitro*, indicating the viral nature of the oncogenic material. The occurrence of R or H virus-like particles, both in tumor cells and in cultures, suggests a probable participation of such structures in the oncogenic mechanisms of the neoplasm. Tumor production was absent after inoculation of acellular suspensions obtained from BHK 21 clone 13 cells or solid s.c. tumor cells using the same experimental procedures as for the ascites tumor. This could be due to variations in the viral agent's characteristics acquired during successive i.p. transfers. Radio-induced immunosuppression of the animals in the course of BHK ascitic tumor production could also account for the suggested virus activity modification.

- 3838 GENETIC FACTORS IN CHRONIC REMITTENT FRIEND DISEASE. (E.) Dawson, P. J. (U. Oregon Med. Sch., Portland) and A. H. Fieldsteel. *Cancer Res* 33(10):2456-2458, 1973.

C57BL/6 and C57BL/Ks mice possess the alleles H-2^b and H-2^d, resp. A study of Friend disease in C57BL/6 x DBA/2 F₁ and C67BL/Ks x DBA/2 F₁ mice showed that remittent disease occurred in the former hybrids and progressive disease in the latter. Since both hybrids have the same Fv-1

and Fv-2 genotypes the change in the character of the disease was ascribed to differences in the Rgv-1 gene located near the H-2 locus. The lymphatic leukemia-inducing virus that acts as helper for Friend virus was shown to replicate better in C57BL/Ks than in C57BL/6 mice, suggesting that Rgv-1 gene may regulate the occurrence of remissions through its action on the helper virus.

- 3839 FELINE VIRUS-INDUCED TUMORS AND THE IMMUNE RESPONSE: RECENT DEVELOPMENTS. (E.) Essex, M. (Harvard U. Sch Public Hlth., Boston, Mass.), S. M. Cotter and J. L. Carpenter. *Am J Vet Res* 34(6):809-812, 1973.

Significant advancements have been made in the last two yr concerning the role of the immune response in virus-induced feline tumors and the natural history of the feline oncornaviruses (FOV). Observations of apparent horizontal transmission have been made in both feral and laboratory cat populations. Salivary excretion and intranasal infection have been demonstrated with the feline leukemia virus (FeLV). It has been shown that cats are not immunologically tolerant to the principal virion antigens as was once thought. Thymic aplasia accompanied by a depression of the cell-mediated immune response has been observed in cats incubating leukemia. New tumor cell membrane antigens have been demonstrated in cells exposed to these viruses. These antigens are also expressed in animals exposed to live virus. High antibody levels to the antigen are associated with protection against the progressive growth of virus-induced sarcomas, and antibody either is absent or present in lower quantities in animals with progressing virus-induced sarcomas. Approximately 13% of 71 healthy adult cats examined in the Boston area had antibody titers to the membrane antigen of 8 or higher. The results reviewed in this article indicate that horizontal transmission should be considered as a significant possibility for the distribution of oncornaviruses among cats under natural conditions.

- 3840 CONTACT-INHIBITED REVERTANT CELL LINES ISOLATED FROM SV40-TRANSFORMED CELLS. V. CONTACT INHIBITION OF SUGAR TRANSPORT. (E.) Schultz, A. R. (Case Western Reserve U., Sch. Med., Cleveland, Ohio) and L. A. Culp. *Exp Cell Res* 81:95-103, 1973.

The transport of 2-deoxyglucose in BALB/c 3T3 cells, Simian virus 40-transformed BALB/c 3T3 (SVT2) cells, and concanavalin A-selected revertant cells of SVT2 was measured. Sparsely-seeded BALB/c 3T3 cells transport the sugar at about 1/4, and sparsely-seeded revertant cells at 3/4, the rate of SVT2 cells. BALB/c 3T3 cells undergo a dramatic drop in sugar uptake at confluency, transporting sugar at about 1/10 the rate of subconfluent cells. Revertant cells (contact-inhibited variants of transformed cells) are similar in this respect, but the drop is only 5-fold. SVT2 cells show no such change in uptake over wide cell densities. Subconfluent BALB/c 3T3, SVT2, and revertant cells have similar K_m and V_{max} values for

2-deoxyglucose transport; however, confluent 3T3 and confluent revertant cells show a large increase in K_m and a 5-fold decrease in V_{max} as compared with their subconfluent counterparts or SVT2 cells--indications of a decreased number of transport sites and a decreased affinity of these sites for sugar when these cells make intimate contacts with each other. These data indicate that extensive changes in the architecture of the cell surface occur when contact-inhibited cells are in close apposition with each other, regardless of the persistence of partially expressed SV40 genetic information. The data also provide support for the hypotheses that transport sites in confluent, contact-inhibited cells are covered by an additional membrane component or changed configurationally. The change in K_m in association with the change in V_{max} suggests a steric change in the membrane of these cells.

- 3841 ANALYSIS OF THE EARLY AND LATE PRODUCTS OF THE DNA POLYMERASE OF FRIEND MURINE LEUKEMIA VIRUS WITH ACTINOMYCIN D. (E.) Swindlehurst, M. (Department Biol., Wesleyan U., Middletown, Conn.). *FEBS Lett* 35(1):24-30, 1973.

The transcription of RNA and DNA in Friend Murine Leukemia virus in the presence of actinomycin D was examined. The rate reaction profiles establish that actinomycin D (30 μ g/ml) retards the transcription of RNA as well as DNA *in vitro*. The product from actinomycin D inhibited reactions is of lower average density and molecular wt than the normal reaction product. The results indicate that only the synthesis of products low in G-C content is insensitive to actinomycin inhibition. In the presence of actinomycin D the Friend virus DNA polymerase might be prevented from copying heteropolymeric regions of the 60-70 S viral RNA. Because of presumed actinomycin binding to the template, the polymerase preferentially copies the more accessible regions of RNA's that have become separated from the bulk of the 60-70 S RNA.

- 3842 VIROGENIC PROPERTIES OF AKR MOUSE SARCOMA CELLS INDUCED BY ROUS SARCOMA VIRUS. (E.) Kuwata, T. (Sch. Med., Chiba U., Japan), T. Okazaki and G. J. Spahn. *Arch Gesamte Virusforsch* 41(1-2): 106-115, 1973.

Sarcomas were induced in AKR mice by Schmidt-Ruppin strain of Rous sarcoma virus (RSV). Sarcoma cells were passaged *in vivo* and *in vitro*, and their virogenicities examined. They did not produce any infectious RSV, but when sarcoma cell suspensions were inoculated into the wing webs of chickens, sarcomas were induced with high frequencies. Moreover, it was verified by physical and biological methods that a clonal line (K3b) of AKR sarcoma cells cultivated *in vitro*, release murine leukemia virus. However, after a long-term cultivation *in vitro*, the sarcoma-inducing capacity of these cells in chickens gradually decreased. The cause of this phenomenon is not clear. These results suggest that RSV and murine leukemia virus genomes coexist in the K3b cells.

- 3843 THE DEFICIENT DENSITY-DEPENDENT GROWTH CONTROL OF HUMAN MALIGNANT GLIOMA CELLS AND VIRUS-TRANSFORMED GLIA-LIKE CELLS IN CULTURE. (E.) Westermarck, B. (Wallenberg Lab., U. Uppsala, Sweden). *Int J Cancer* 12(2):438-451, 1973.

The density-dependent growth control of eight established human glioma lines and of human glia-like cells transformed by simian virus 40 and feline sarcoma virus was investigated *in vitro*. All neoplastic lines reached higher terminal cell densities ($170-600 \times 10^3$ cells/cm²) than the corresponding normal glia-like cells ($50-90 \times 10^3$ cells/cm²). A marked decrease in proliferation rate occurred at high cell densities which could not be explained by medium depletion or accumulation of toxic substances. An absolute resting phase was not reached by the neoplastic cells; a considerable residual DNA synthesis continued even at the highest cell density levels which could be reached. The intervals of the cell cycle were determined on one glioma line. Cells inhibited in crowded cultures were arrested in G₁. The results imply that at least a proportion of neoplastic human glia lines retain a degree of topoinhibition, i.e., are still sensitive to growth restraint induced by increased cell density. However, topoinhibition was never as complete as among normal cells, since possibly cell density-independent ("autonomous") multiplication was maintained at a fairly high rate even at the abnormally high cell densities which all neoplastic lines attained.

- 3844 TWO DISTINCT TYPES OF ENHANCEMENT OF GALACTOSE UPTAKE INTO HAMSTER CELLS: TUMOR-VIRUS TRANSFORMATION AND HEXOSE STARVATION. (E.) Kalckar, H. M. (Harvard Med. Sch., Boston, Mass.) and D. Ullrey. *Proc Natl Acad Sci USA* 70(9):2502-2504, 1973.

Enhancement of hexose uptake and enhancement of sugar uptake can clearly be demonstrated in cultures of hamster cells when uptake of ¹⁴C-labeled galactose is monitored after 10 or 20 min. The profiles of accumulation products are strikingly different under various conditions of enhancement. In cultures of hamster NIL cells transformed with polyoma virus, much of the ¹⁴C is accumulated as uridinediphosphohexose (UDPhexose). Untransformed cells accumulate galactose-1-phosphate as well as UDPhexose. Hexose-starved cells show enhanced uptake of galactose; however, this marked enhancement was only observed in NIL cultures close to contact inhibition. The novel and common feature seen in hexose-starved cells when incubated briefly with ¹⁴C-labeled galactose is the occurrence of a marked accumulation of [¹⁴C]UDPhexuronic acid at the expense of UDPhexose. The ratio [¹⁴C]UDPhexuronic acid/UDPhexose in cultures fed glucose or galactose was invariably low (0.15-0.2) regardless of the presence or absence of contact inhibition. Hexose starvation of 20 hr invariably changed this ratio by a factor of 10 or more, due to accumulation of UDPhexuronic acid. This result was also observed in cultures transformed with polyoma virus. The presence of 3-O-methylglucose in the growth medium did not alter the typical "sugar starvation pattern" (i.e., the UDPhexuronic acid/UDPhexose ratio averaged 1.7). Enhancement of galactose

uptake by hexose starvation was very pronounced in NIL cultures that were close to contact inhibition, but was not a prominent feature in the polyoma-transformed cultures. The transformed cells grown on glucose or galactose growth medium showed the usual enhanced rate of uptake of galactose as compared with nontransformed near-confluent cultures that had been fed hexose. The polyoma-induced enhancement showed none of the features characteristic of hexose-starved cells.

- 3845 CHARACTERIZATION OF THE LOW-MOLECULAR-WEIGHT RNAs ASSOCIATED WITH THE 70S RNA OF ROUS SARCOMA VIRUS. (E.) Faras, A. J. (Dept. Microbiol., U. California, San Francisco), A. C. Garapin, W. E. Levinson, J. M. Bishop and H. M. Goodman. *J Virol* 12(2):334-342, 1973.

Two low-molecular-wt RNAs are associated with the 70S RNA complex of Rous sarcoma virus: a previously described 4S RNA and a newly identified 5S RNA. The 4S RNA constitutes 3-4% of the 70S RNA complex or the equivalent of 12-20 molecules per 70S RNA. It exhibits a number of structural properties characteristic of transfer RNA as revealed by two-dimensional electrophoresis of oligonucleotides obtained from a T1 ribonuclease digest of the 4S RNA species. The 5S RNA is approximately 120 nucleotides in length, constitutes 1% of the 70S RNA complex or the equivalent of 3-4 molecules/molecule of 70S RNA, and is identical in nucleotide composition and structure to 5S RNA from uninfected chicken embryo fibroblasts. Melting studies indicate that the 5S RNA is released from the 70S RNA complex at the same temperature required to dissociate 70S RNA into its constituent 35S subunits. In contrast, greater than 80% of the 4S RNA is released from 70S RNA prior to its conversion into subunits. The possible biological significance of these 70S-associated RNAs is discussed.

- 3846 NUCLEOPROTEIN COMPLEXES CONTAINING REPLICATING SIMIAN VIRUS 40 DNA: COMPARISON WITH POLYOMA NUCLEOPROTEIN COMPLEXES. (E.) Hall, M. R. (Scripps Clinic Res. Fdn., La Jolla, Calif.), W. Meinke and D. A. Goldstein. *J Virol* 12(4):901-908, 1973.

Procedures for isolating nucleoprotein complexes containing replicating polyoma DNA from infected mouse cells were used to prepare short-lived nucleoprotein complexes (r-SV40 complexes) containing replicating simian virus 40 (SV40) DNA from infected monkey cells. Like the polyoma complexes, r-SV40 complexes were only partially released from nuclei by cell lysis but could be extracted from nuclei by prolonged treatment with solutions containing Triton X-100. r-SV40 complexes sedimented faster than complexes containing SV40 supercoiled DNA (SV40 complex) in sucrose gradients, and both types of SV40 nucleoprotein complexes sedimented ahead of polyoma complexes containing supercoiled polyoma DNA (py complex). The sedimentation rates of py complex and SV40 complex were 56 and 61S, resp., based on the sedimentation rate of the mouse large ribosomal subunit as a marker, r-SV40 complexes sedimented as multiple peaks between 56 and

75S. Sedimentation and buoyant density measurements indicated that protein is bound to all forms of SV40 DNA at about the same ratio of protein to DNA (1-2/1) as was reported for polyoma nucleoproteins.

- 3847 HERPESVIRUS AND CERVICAL CANCER. EPILOG. (E.) Deinhardt, F. (Rush-Presbyterian-St. Luke's Med. Ctr., Chicago, Ill.). *Cancer Res* 33(6):1556, 1973.

The Organizing Committee of the American Cancer Society's Symposium on herpesvirus and cervical cancer, December, 1972, summarizes ideas presented for clarifying the role of herpesvirus in neoplasia, particularly of herpes simplex type 2 and cervical carcinoma. It was suggested that tools for differential diagnosis of herpesvirus infections and tests for identification of herpes type 1 and type 2 strains be developed, with the aid of a small central work group. The exact antigenic identity of the world's herpesvirus strains should be established. A prospective epidemiological study considering more than one etiological factor in multivariate analysis should be conducted, and coordinated along with ongoing related studies. Basic research and collaborative studies on the virology of cervical carcinoma and other neoplasias should be encouraged, and studies of the oncogenicity of herpesvirus *in vivo* and *in vitro* given high priority. More fundamental studies on the immunology of acute, chronic and "tumorigenic (?)" herpesvirus infections are urged and suggestions made regarding their content. The committee stressed the value of interdisciplinary and well coordinated studies in all of these areas.

- 3848 LEUKEMIA-SPECIFIC DNA SEQUENCES IN LEUKOCYTES OF THE LEUKEMIC MEMBER OF IDENTICAL TWINS. (E.) Baxt, W. (Coll. Physicians Surg., Columbia U., New York, N.Y.), J. W. Yates, H. J. Wallace, Jr., J. F. Holland and S. Spiegelman. *Proc Natl Acad Sci USA* 70(9):2629-2632, 1973.

The discovery in human leukemic cells of particulate elements encapsulating 70S RNA and RNA-directed DNA polymerase made possible the synthesis of a [³H]DNA probe that could detect leukemia-specific sequences in the DNA of normal and leukemic individuals. An earlier study of a series of unrelated leukemic patients established that the nuclear DNA of their leukemic cells contain particle-related sequences that cannot be detected in leukocytes of normal individuals. This result was inconsistent with the virogene concept that demands the inclusion of one complete copy of oncogenic information in the genome of every normal cell. The same difference between leukemic patients and normal individuals was found in the present study of two sets of identical twins, each set containing a member with acute myelotic leukemia. In each pair, the leukemic member contained particle-related sequences in the DNA of his leukocytes that could not be detected in the leukocytes of his healthy identical sibling. This finding implies that the additional leukemia-specific information found in the DNA of the leukemic individuals must have been inserted subsequent to fertilization. This outcome argues against the virogene hypothesis or any other

etiologic concept that invokes vertical transmission through the germ line of the particle-related information found uniquely in the DNA of leukemic cells.

- 3849 TRANSFORMATION OF MOUSE CELLS INFECTED WITH AKR LEUKEMIA VIRUS BY BENZENE EXTRACT FRACTIONS OF CITY AIR PARTICLES. (E.) Rhim, J. S. (Microbiol. Assoc., Inc., Bethesda, Md.), R. J. Gordon, R. J. Bryan and R. J. Huebner. *Int J Cancer* 12(2):485-492, 1973.

Fractions of benzene extract of particles from city air were studied for their transforming activity in the AKR leukemia virus-infected NIH Swiss mouse embryo (AKR-NIH-ME) cell system. Mouse embryo cells chronically infected with AKR leukemia virus were transformed by 1.0 or 0.1 µg/ml benzene smog extracts, most of its fractions, and certain chemicals, whereas uninfected mouse embryo cultures were not transformed. Most of the transformed cells produced tumors when transplanted (10^6 cells/animal, s.c.) into newborn NIH Swiss mice. All tumors were sarcomas. The three neutral fractions, AE₀S₂ (tetracyclics), AE₀S₃ (pentacyclics, including benzo(a)pyrene), and AE₀S₅ (unknown structural type), and two acid, phenol fractions, AE₄ and AE₄B, had the strongest transforming activity *in vitro*. One fraction, AE₁B (CHCl₂ back extract of water washes from AE1) appeared to be inactive in the *in vitro* system at the concentrations tested.

- 3850 LACK OF SEQUENCE HOMOLOGY BETWEEN THE 70S RNA OF VARIOUS RNA TUMOR VIRUSES AND THE DNA OF SIMIAN VIRUS 40 OR POLYOMA VIRUS. (E.) Gallagher, R. E. (Natl. Cancer Inst., Bethesda, Md.), A. S. Levine, D. H. Gillespie and R. C. Gallo. *J Virol* 11(6):1027-1029, 1973.

No significant hybridization was detected of DNA from simian virus 40 or polyoma virus, and of ³H-70S RNA from avian myeloblastosis virus, murine leukemia virus (Rauscher), murine sarcoma virus (Kirsten), RD-114B, simian sarcoma virus-1, or Mason-Pfizer virus. Based on ³H-70S RNA specific activity, estimated by a ³H-polyuridylic acid method, and net counts/min of ³H-70S RNA bound to DNA, there is a lack of sequence homology between the nucleic acids of the tested RNA and DNA tumor viruses.

- 3851 CHARACTERISTICS OF MURINE C-TYPE VIRUSES. I. INDEPENDENT ASSORTMENT OF INFECTIVITY IN ONE *IN VIVO* AND FOUR *IN VITRO* ASSAYS. (E.) Fenyö, E. M. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden) and G. Grander. *Int J Cancer* 12(2):452-462, 1973.

The infective properties of mouse C-type viruses produced by a Maloney leukemia virus-induced lymphoma line (YAC) and its immunoresistant subline (YACIR), mouse L cells (A9 fibroblast line and its malignant derivative, A9HT), and their hybrids were investigated in one *in vivo* and four *in vitro* assays. While the two lymphoma-derived viruses differed in their ability to convert antigenically JLS-V9 cells, they were

equally efficient in the XC-plaque test. The same viruses also transformed BALB/3T3 cells, similarly to the virus produced by A9 cells. On the other hand, transformation by the A9 virus was not accompanied by plaque formation on XC cells (except distinct single giant-cell formation). The reverse situation, positive plaque test without transformation, was also encountered (YACIR-A9 virus). Whether plaque forming, antigen inducing, or transforming, all viruses were immunogenic. The results indicate that virus released by a given cell line may be detected by one test and not by the other. The characteristic infectivity pattern of a virus produced by a certain cell line remained stable over a period of 7 months.

- 3852 FLUORESCENCE STUDIES ON POLYOMA VIRUS. (E.) Kondo, M. (Res. Inst., Microbial Dis., Osaka U., Japan), M. Takeuchi, H. Nomaguchi and J. Kamahora. *Biken J* 16(2):31-38, 1973.

Polyoma virus showed fluorescence with a max at 333 mµ when irradiated with UV light. When various denaturing agents, such as NaOH, HCl, urea, dichloroacetic acid, and guanidine-HCl, were added to polyoma virus, the fluorescence intensity of the virus decreased with decrease of hemagglutination activity. Fluorescence emission spectra were used to investigate conformational changes in the virus protein caused by denaturing agents. The fluorescence of polyoma virus seems to be due to the tryptophan residues in the virus protein for the following reasons: 1) the fluorescence spectrum of the native virus coincides with the spectrum of tryptophan in hydrophobic medium; 2) the fluorescence spectrum of the strongly denatured virus coincides with the spectrum of tryptophan in a polar medium; 3) the peak wavelength of the action spectrum of loss of fluorescence intensity coincides with the absorption wavelength of tryptophan; 4) the fluorescence spectrum of proteins containing tryptophan only shows the fluorescence of this residue; and 5) DNA and lipid have negligible fluorescence.

- 3853 ULTRASTRUCTURAL COMPARISON OF TRANSFORMED HUMAN CELLS AND THEIR NORMAL COUNTERPARTS AT VARIOUS PASSAGE LEVELS. (E.) Lipetz, J. (Dept. Biol. Sci., Drexel U., Philadelphia, Pa.). *J Ultrastruc Res* 44(1-2):1-10, 1973.

A comparison of the ultrastructures of two lines of human cells transformed by the oncogenic virus, SV40, revealed that the percentage of mitochondria with completely transverse cristae, the number of lysosomes and autophagic vacuoles, the morphology of the nucleus and endoplasmic reticulum, and the paucity of Golgi apparatus, were similar in both lines. These characteristics, previously shown to be age-related in the cells from which these lines originated, are similar to those found in young normal cells. These transformed lines differed from each other only in their percentage of bizarre mitochondria. Since one of the transformed lines originated from cells in early passage and the other from cells in late passage, the inverse correlation between Golgi-lysosomal activity and division potential suggests that the line derived from late passage cells either underwent a major change

in this activity or is derived from the occasional "young" cells believed to occur in old cultures.

- 3854 INDUCTION OF TUMORS IN HAMSTERS WITH A BOVINE ADENOVIRUS STRAIN (SEROTYPE 8). (E.) Rondhuis, P. R. (Central Vet. Inst., Rotterdam, Netherlands). *Archiv Gesamte Virusforsch* 41(1-2):147-149, 1973.

Several litters of hamsters were inoculated s.c. on the day of birth with type 8 strain 6833 bovine adenovirus. Hamsters in the control group were inoculated with material from uninfected calf cell cultures. During the 21-month observation period, 40 of the 56 hamsters in the virus group developed one or more s.c. tumors while none developed in the 51 hamsters in the control group. The greater number of tumors developed 5-15 months after inoculation, and varied in size from 5-50 mm in diameter. The tumors were of the fibrosarcoma type. Signs of malignancy included: large pleomorphic nuclei with distinct nucleoli, high mitotic activity, hemorrhage and necrosis.

- 3855 THE EFFECT OF CORDYCEPIN ON CELL TRANSFORMATION BY RNA TUMOR VIRUSES. (E.) Lovinger, G. G. (Flow Lab., Inc., Rockville, Md.), R. A. Klein, R. V. Gilden and M. Hatanaka. *Virology* 55(2):524-526, 1973.

The transformation of BALB mouse embryo fibroblasts by the Rauscher pseudotype of the Moloney strain of murine sarcoma virus was inhibited by cordycepin (3'-deoxyadenosine, 25 µg/ml). The drug was most inhibitory when added 30 to 90 minutes after infection. Adenosine, but not 2'-deoxyadenosine, reversed the inhibitory effect. Several possible mechanisms of action of the drug are discussed. If phosphorylated to the triphosphate level, cordycepin may inhibit an ATP-dependent enzyme essential for fixation of transformation. Alternatively, cordycepin triphosphate may be reduced in the 2' position of the ribose moiety, enter the DNA precursor pool, and inhibit viral DNA synthesis. The drug could also act by depressing RNA synthesis in the early stages of infection.

- 3856 STUDIES OF NONDEFECTIVE ADENOVIRUS 2-SIMIAN VIRUS 40 HYBRID VIRUSES. VIII. ASSOCIATION OF SIMIAN VIRUS 40 TRANSPLANTATION ANTIGEN WITH A SPECIFIC REGION OF THE EARLY VIRAL GENOME. (E.) Lewis, A. M., Jr. (Natl. Inst. Allergy Infect. Dis., Bethesda, Md.) and W. P. Rowe. *J Virol* 12(4):836-840, 1973.

Two of the five nondefective adenovirus 2 (Ad2)-simian virus 40 (SV40) hybrids induce SV40 transplantation resistance in immunized hamsters. These two hybrids, Ad2+ND₂ and Ad2+ND₄, were originally isolated from human embryonic kidney cells and African green monkey kidney cells, resp.; they contain 32 and 43% of the SV40 genome, resp. Immunization i.p. with the hybrids resulted in a 13- to 20-fold increase in hamster resistance to tumor induction by SV40-transformed hamster kidney cells injected s.c. The pattern of induction of SV40 transplantation antigen (TSTA) by the various hybrids differentiates TSTA from both SV40 U

and T antigens. Since the SV40 RNA induced by both these hybrids is early SV40 RNA, these findings confirm that TSTA is an early SV40 function. By combining available data on SV40 antigen induction by these hybrids with electron microscopy heteroduplex mapping studies, the DNA segment responsible for the induction of SV40 TSTA can be inferred to lie in the region between 0.17 and 0.43 SV40 units from the site on the SV40 chromosome cleaved by *E. coli* R₁ restriction endonuclease.

- 3857 INDUCTION OF TYPE C VIRUSES IN CULTURED GUINEA PIG CELLS. (E.) Nayak, D. P. (Sch. Med., U. California, Los Angeles) and P. R. Murray. *J Virol* 12(1):177-187, 1973.

Particles morphologically resembling type C viruses were activated by bromodeoxyuridine (BUDR; 10⁻⁴ M) treatment of cultured guinea pig cells. Virus particles were isolated from the cells of normal and leukemic strain 2 and random-bred guinea pigs (adult and embryonic). Immature virus particles with electron-lucent cores were found in the cytoplasmic matrix. The mature particles with electron-dense cores were found outside the cells and some appeared in the process of budding from the plasma membrane. The peak of virus production was observed within 4 days of BUDR treatment. When compared with the amount of virus produced in darkness, visible light enhanced virus production, whereas exposure of BUDR-treated cells to UV (4 ergs/mm²/sec) light either had no effect or inhibited virus production. Virus particles had a density of 1.16 g/ml, possessed an oncornavirus-specific reverse transcriptase, and contained a large-molecular-wt RNA (65-70S) which dissociated into 36S subunits after heat denaturation. The BUDR-activated virus particles, therefore, possessed the morphological, biophysical, and biochemical characteristics of type C oncornaviruses.

- 3858 INFECTIOUS TYPE C VIRUSES RELEASED BY NORMAL CAT EMBRYO CELLS. (E.) Todaro, G. J. (Natl. Cancer Inst., Bethesda, Md.), R. E. Benveniste, M. M. Lieber and D. M. Livingston. *Virology* 55(2):506-515, 1973.

Type C viruses with antigenic and host range properties similar to the RD114 virus and to a virus from a continuous line of cat kidney fibroblast cells, CCC, were isolated from six of ten diploid fetal cat cell cultures. Two strains spontaneously produce high virus levels, and the virus can be visualized by electron microscopy. The other four lines yield infectious virus upon cocultivation with a permissive host cell. Infectious type C viruses were found in cultures derived from kidney, thymus, and lung, as well as from whole embryo cultures. Each of the new viruses replicates readily in human and rhesus monkey cells, but not in most other cat cell strains tested. However, one cat cell strain, FFc60WF, is permissive for replication of the RD/CCC group of type C viruses produced by other cat cell strains. The host range of these endogenous type C viruses includes bat, dog, mink, and rabbit cells, as well as primate cells. Type C viruses of the FeLV group

could not be detected in any of these ten fetal cat cell cultures. Viral RNA from one of the new isolates, from the CCC virus, and from RD114 virus each specifically anneal S_1 to an [3H]DNA transcript of the RD114 genome.

- 3859 PARTIAL TRANSCRIPTION OF MURINE TYPE C VIRAL GENOMES IN BALB/c CELL LINES. (E.) Benveniste, R. E. (Nat'l. Cancer Inst., Bethesda, Md.), G. T. Todaro E. M. Scolnick and W. P. Parks. *J Virol* 12(4):711-720, 1973.

The mouse cell line BALB/c 3T3 and its derivatives transformed spontaneously or by treatment with a variety of external agents were analyzed for cytoplasmic RNA complementary to DNA products prepared from the Kirsten strain of murine sarcoma-leukemia virus, and from an endogenous type C virus of BALB/c 3T3. Although none of these cell lines spontaneously releases complete type C virions, they all contain RNA which is partially homologous to a portion of the 35S RNA isolated from these viruses. The parental cell line, BALB/c 3T3, contains a low level of viral-related RNA, and there is an increased amount of this RNA in some of the transformed cells. The RNA detected represents only a fraction of the viral RNA found in virus-producing cells. The formation of RNA:DNA hybrids was detected by equilibrium centrifugation in Cs_2SO_4 density gradients and by analysis with a single-strand-specific nuclease from *Aspergillus oryzae*. Viral DNA products prepared from an endogenous reaction with whole virus in the presence of actinomycin D or from purified 70S viral RNA as template using avian myeloblastosis virus DNA polymerase yield comparable data. In addition, all of the BALB/c lines examined produce detectable levels of murine type C virus group-specific antigen.

- 3860 INTRACELLULAR FORMS OF ADENOVIRUS DNA. II. ISOLATION IN DYE-BUOYANT DENSITY GRADIENTS OF A DNA-RNA COMPLEX FROM KB CELLS INFECTED WITH ADENOVIRUS TYPE 2. (E.) Doerfler, W. (Rockefeller U., New York, N.Y.), U. Lundholm, U. Rensing and L. Philipson. *J Virol* 12(4):793-807, 1973.

DNA-RNA complexes with different DNA/RNA ratios were isolated from KB cells productively infected with human adenovirus type 2 by the dye-buoyant density procedure by using propidium iodide. Both the DNA and RNA components of these complexes are virus specific, as shown by nucleic acid annealing experiments, and parental as well as newly synthesized viral DNA can be recovered from these complexes. The RNA component is susceptible to digestion with pancreatic ribonuclease at low and high salt concentrations. The RNA is in part liberated from the complex during purification over several cycles of equilibrium centrifugation in dye-buoyant density and Cs_2SO_4 gradients. Analysis in the latter gradients reveals at least four classes of virus-specific nucleic acid: (i) RNA loosely bound and released during purification, (ii) and (iii) two distinct classes of RNA-DNA complexes with different DNA/RNA ratios, and (iv) DNA apparently not associated with RNA. The RNA component in the DNA-RNA complexes is as large as 1.6×10^6 daltons. The biological function of these complexes is still uncertain, but they may be transcription complexes.

- 3861 PROCESSING OF ADENOVIRUS 2-INDUCED PROTEINS. (E.) Anderson, C. W. (Cold Spring Harbor Lab., N.Y.), P. R. Baum and R. F. Gesteland. *J Virol* 12(2):241-252, 1973.

Analysis of ^{35}S -methionine-labeled extracts of adenovirus 2-infected KB cells revealed 22 virus-induced polypeptide components. The peptides were detected by autoradiography after electrophoresis on sodium dodecyl sulfate-polyacrylamide gels. Most proteins of the virion were easily detected in extracts of whole cells labeled for short periods between 15 and 30 hr after infection; however, several virion components were conspicuously absent. Radioactivity appeared in two of these virion components during a chase in nonradioactive medium, and this appearance was paralleled by a decrease in the radioactivity associated with two nonvirion adenovirus-induced proteins, results which imply precursor-product relationships for these components. Comparison of one of the chasable adenovirus-induced components (designated P-VII; mass of 20,000 daltons) and the major core protein (VII; mass of 18,500 daltons) of the virion showed that they have four common methionine-containing tryptic peptides; P-VII has an additional methionine residue which is not found in the major core protein. It is proposed that at least two of the adenovirus 2 virion components are derived by the cleavage of higher molecular wt precursor polypeptides.

- 3862 TRANSFORMATION OF MAMMALIAN CELLS BY DNA-CONTAINING VIRUSES FOLLOWING PHOTODYNAMIC INACTIVATION. (E.) Rapp, F. (Milton S. Hershey Med. Ctr., Hershey, Pa.), J.-L. H. Li and M. Jerkofsky. *Virology* 55(2):339-346, 1973.

Simian papovavirus-40 (SV40) and herpes simplex virus (HSV) types 1 and 2 were inactivated by exposure to fluorescent light after replication in cells pretreated with neutral red. Exposure of hamster embryo fibroblast cells to appropriately inactivated virus yielded clones of cells showing loss of contact inhibition. Cell lines were established from clones arising from "transformation" by all viruses tested. SV40-transformed cell lines were more epithelioid than the corresponding control cells. Mononucleate and multinucleate cells contained multiple prominent nuclei. In HSV-1- and HSV-2-transformed cell lines, most cells were fibroblastic; a few cells were epithelioid and there were some multinucleated cells. SV40 tumor (T) antigen was detected in the nuclei of the SV40-transformed cells and herpes simplex virus specific antigens were demonstrated in the cytoplasm of both type 1- and type 2-transformed cells. Virus was not recovered from the transformed cells. The results indicate that DNA-containing viruses are able to transform mammalian cells after photodynamic inactivation of infectivity.

- 3863 INTEGRATED STATE OF ONCORNAVIRUS DNA IN NORMAL CHICKEN CELLS AND IN CELLS TRANSFORMED BY AVIAN MYELOBLASTOSIS VIRUS. (E.) Markham, P. D. (U. California Sch. Med., Los Angeles) and M. A. Baluda. *J Virol* 12(4):721-732, 1973.

The covalent linkage of oncornavirus-specific DNA to

chicken DNA was investigated in normal chicken embryo fibroblasts (CEF) and in virus-producing leukemic cells transformed by avian myeloblastosis virus (AMV). The virus-specific sequences present in cellular DNA fractionated by different methods were detected by DNA-RNA hybridization by using 70S AMV RNA as a probe. In CEF and in leukemic cells, the viral DNA appeared to be present only in the nucleus. After cesium chloride-ethidium bromide density equilibrium sedimentation, the viral DNA was present as linear, double-stranded molecules not separable from linear chicken DNA. After extraction by the Hirt procedure, the viral DNA precipitated with the high-molecular-wt DNA. After alkaline sucrose velocity sedimentation, the viral DNA cosedimented with the high-molecular-wt cellular DNA. The results indicate that in both types of cells studied, the oncornavirus-specific DNA sequences were covalently linked by alkali stable bonds to nuclear cellular DNA of high molecular wt and were not present in free form of any size.

3864 UNWINDING OF PARENTAL STRANDS DURING
SIMIAN VIRUS 40 DNA REPLICATION. (E.)

Salzman, N. P. (Natl. Inst. Allergy Infect. Dis., Bethesda, Md.), E. D. Sebring and M. Radonovich. *J Virol* 12(4):669-676, 1973.

Pools of young (less than 60% replicated) and mature (60-90% replicated) replicating molecules of simian virus 40 (SV40) DNA were treated at pH 12.2 to dissociate growing chains from the parental strands. The molecules were neutralized so that the parental strands could reassociate and they were then isolated. They are covalently closed structures which sediment rapidly in alkaline sucrose gradients; however, the sedimentation rates are less than the sedimentation rate of SV40 DNA I. Isopycnic banding in CsCl-ethidium bromide and sedimentation velocity studies in the presence of various amounts of ethidium bromide indicate that these structures contain negative superhelical turns and several-fold higher superhelix densities than SV40 DNA I (the covalently closed DNA molecule). These structures are those that would be predicted if nicking, unwinding, and sealing of the parental strands occurred as replication proceeded. These experiments provide a direct demonstration that there is a progressive decrease in the topological winding number which accompanies SV40 DNA replication.

3865 PROPERTIES OF NUCLEOPROTEIN COMPLEXES CONTAINING REPLICATING POLYOMA DNA. (E.) Goldstein, D. A. (Scripps Clin. Res. Fdn., La Jolla, Calif.) M. R. Hall and W. Meinke. *J Virol* 12(4):887-900, 1973.

Short-lived nucleoprotein complexes (r-py complex) containing replicating polyoma DNA were isolated from infected cells after lysis with Triton X-100. The Triton lysing procedure releases most complexes containing supercoiled viral DNA (py complex) from nuclei, but liberates only a portion of r-py complexes. The latter complexes are associated more strongly with nuclear sites but can be extracted by prolonged incubation of nuclei in lysing solution. Complexes containing replicating polyoma DNA appear to be precursors to sta-

ble complexes containing supercoiled DNA. Sedimentation and buoyant density studies indicate that protein is bound to both r-py complexes and py complexes at a ratio of protein to DNA of about 1 to 2/1. Both types of complexes sediment as if the viral DNA is more compact than free DNA and both undergo major reversible configurational changes with increased salt concentration. Changes resulting from enzymatic and chemical treatment indicate that there may be two or more protein components in both r-py complex and py complex. One component is digested by Pronase and trypsin while another is resistant to the enzymes but released by deoxycholate. The abundance and similarity in chemical and physical properties of protein bound to all forms of polyoma DNA suggest that part of the protein molecules may serve in a structural capacity.

3866 ADAPTATION OF PLAQUE ASSAY METHODS TO THE
IN VITRO QUANTITATION OF THE RADIATION
LEUKEMIA VIRUS. (E.) Niwa, O. (Stanford U. Sch. Med., Calif.), A. Decleve, M. Lieberman and H. S. Kaplan. *J Virol* 12(1):68-73, 1973.

A modification of the XC cell procedure for murine leukemia virus assay which yields quantitative data over a wide range of virus concentrations is described. By using serial passage of infected cell cultures and reversal of the plating sequence in the XC procedure, titers of radiation leukemia virus (RadLV) were obtained, from a continuous line of C57BL/Ka mouse embryo fibroblast cells, which were about 10-fold higher than those found by using the conventional assay. The end-point dilution titer of the *in vitro*-derived virus was consistently in the range 5×10^7 to 5×10^8 plaque-forming U/ml. By using the modified procedure, it was observed that, even at high multiplicities of infection, less than 10% of the cells function as infective centers, although the proportion increases with serial passage. It was also observed that exposure of infected cells to UV light (1600 ergs/mm²), which is commonly used to make plaques more visible in the conventional XC cell test, inhibits plaque formation in the RadLV system. Substitution of X irradiation (10 Krads) for UV exposure improved plaque visibility without loss of sensitivity.

3867 BURKITT'S LYMPHOMA AND HEREDITY. (E.) Innis, M. D. (Princess Alexandra Hosp., Brisbane, Australia). *Oncology* 28(2):184-192, 1973.

In previous reports by the author, evidence was presented to show that the common malignancies of childhood may be heritable disorders controlled by genes in Hardy-Weinberg equilibrium. The Hardy-Weinberg law states that given a stable gene pool, the alleles or alternative expressions of the gene at a particular gene locus will maintain a constant proportion to one another in the population from one period to the next, the precise proportions being a function of the race or ethnic group. Furthermore, it has been shown that nephroblastomas are possibly index cancers which do not vary in relative frequency among different ethnic groups. It is argued that, if Burkitt's lymphoma is also a heritable disorder in

Hardy-Weinberg equilibrium with other malignancies, it should show the same equilibrium with nephroblastomas as do other malignancies, i.e. the relative frequency of nephroblastomas should be the same in Nigeria and Uganda as anywhere else in the world; Nigeria and Uganda being areas in which Burkitt's lymphoma may form about 50% of all childhood neoplasms. A comparison of the relative frequency of nephroblastoma in these areas in fact shows it is not different from any other part of the world. The conclusion is that Burkitt's lymphoma may also be a heritable disorder and the high incidence merely reflects the racial genetic make-up of the people in these areas.

- 3868 PRESENCE OF A THERMOSENSITIVE STEP IN THE COURSE OF TRANSFORMATION BY SE-POLYOMA VIRUS (E.) Hakura, A. (Res. Inst. Microbial Dis., Osaka U., Japan) and Y. Okada. *Virology* 55(2):527-529, 1973.

Wild-type SE-polyoma virus shows a temperature-sensitive phase for transformation of BHK21 cells in soft agar. Suspended BHK21 cells were infected with virus at a multiplicity of 50 plaque-forming U/cell, diluted, and layered on soft agar plates at a concentration of 3.0×10^4 cells/plate. The numbers of transformed colonies were markedly higher following incubation at 35 C compared with 39 C. "Shift up" and "shift down" experiments indicated that the temperature-sensitive step was transient and restricted to the first 48 hr after incubation.

- 3869 PROPERTIES OF NONINFECTIOUS AND TRANSFORMING VIRUSES RELEASED BY MURINE SARCOMA VIRUS-INDUCED HAMSTER TUMOR CELLS. (E.) Gazdar, A. F. (Natl. Cancer Inst., Bethesda, Md.), E. Russell, P. S. Sarma, P. S. Sarin, W. Hall and H. C. Chopra. *J Virol* 12(4):931-936, 1973.

The cell culture lines HTG2 and HTG3 were established from a transplantable hamster tumor induced by a murine sarcoma virus (strain Gz-MSV) after 17 and 60 *in vivo* passages, resp. The viruses released by these two cell lines differ markedly in morphology, antigenic composition, infectivity, transforming ability, and enzymatic activity. HTG2 virions contained the sarcoma genome but were noninfectious for mouse and hamster cells (S+M- virus). HTG3 virions transformed hamster but not mouse cells. Whereas HTG2 cells and virus contained murine type C virus gs-1 antigen, all HTG3 clonal lines expressed both murine and hamster type C virus gs-1 antigens. The RNA-dependent DNA polymerase activity of HTG2 virus was very low, while that of HTG3 virus was relatively high. HTG2 virions contained electron-lucent centers only. HTG3 virus consisted of the expected mixture of virions with electron-dense and electron-lucent centers. Many broken or incomplete virions were present in both viruses. HTG2 virus is a noninfectious "defective" sarcoma virus without detectable helper virus. Data obtained in these experiments suggest that HTG3 virus is a hamster type C virus pseudotype of Gz-MSV (Gz-MSV[HaLV]). The genome of Gz-MSV is capable of antigenic expression in heterologous cells and in the presence of heterologous viruses. Attempts to chemically activate hamster type C virus (HaLV) from HTG2 cells

were unsuccessful. The HTG1 cell culture line, established from another Gz-MSV-induced hamster tumor, initially released a virus indistinguishable from the HTG2 virus. After *in vitro* passage, spontaneous activation of HaLV occurred in HTG1 cells, and the resultant Gz-MSV (HaLV) had properties similar to those of the HTG3 virus.

- 3870 HERPES SIMPLEX VIRUS AND HUMAN CYTOMEGALOVIRUS REPLICATION IN WI-38 CELLS. I. SEQUENCE OF VIRAL REPLICATION. (E.) Smith, J. D. (Cornell U. Grad. Sch. Med. Sci., New York, N.Y.) and E. de Harven. *J Virol* 12(4):919-930, 1973.

A comparison, under standardized conditions in the same host WI-38 cell system, of herpes simplex virus (HSV) and human cytomegalovirus (CMV) revealed differences in viral morphology, in the timing of their infectious cycles, and in several morphological events during those cycles. Structural distinctions between the two viruses included the coating of unenveloped cytoplasmic CMV capsids, but not those of HSV, and a variation in the structure of their cores. Since the two cycles were carried out in the same host cell strain under conditions of one-step growth (input multiplicity = 10 plaque-forming U/cell), it was possible to construct time scales locating the major events of each cycle. Comparison of the two showed that HSV replicated and released progeny within 8 hr postinfection, whereas CMV required 4 days. These results correlated well with those of concurrent plaque assays. Within the longer CMV cycle, most of the major events appeared retarded to a similar degree, and no obvious limiting step in particle production could be identified. Distinctions between the two cycles included the following: condensation of the chromatin in HSV but not CMV-infected cells; the greater tendency of HSV to produce membrane alterations; and the appearance of cytoplasmic dense bodies in CMV- but not HSV-infected cells. Identification of these differences even under identical conditions of culture and infection strongly implies that they result from intrinsic differences in the nature of the viruses, and are not caused by variations in experimental conditions.

- 3871 MECHANISM OF ONCOGENIC TRANSFORMATION BY ROUS SARCOMA VIRUS. III. ROLE OF PROVIRAL DNA IN MORPHOLOGIC CONVERSION OF CHICKEN EMBRYO FIBROBLASTS. (E.) Balduzzi, P. C. (U. Rochester Sch. Med. Dentistry, N.Y.). *J Virol* 12(2):284-290, 1973.

Multiplication of Rous sarcoma virus and morphological conversion of chicken embryo fibroblasts are mediated by a DNA provirus. The role of the provirus in induction of morphological conversion has been shown by experiments of light inactivation of bromodeoxyuridine (BUDR)-sensitized proviral DNA. In the experiments reported here, inactivation of focus formation by BUDR and light could be obtained in cells in which the ability to produce virus has become resistant to X irradiation. Three days after infection of cells with 100 focus-forming units of virus in medium containing 10^{-5} BUDR, X-irradiation (2800 R) resulted in about 50% suppression of focus formation. The proportion of cells releasing virus by day 3

correlated well with the number of foci surviving light exposure. The inactivation of focus formation is considered to reflect the integrated state of the provirus. These experiments indicate that the role of proviral DNA extends beyond induction of morphological conversion and that an intact provirus is required for the maintenance of the transformed state. These experiments also indicate that no irreversible process leading to morphological conversion is initiated by a nonintegrated or by an integrated provirus.

3872 RELATIONSHIP BETWEEN REPLICATION OF SIMIAN VIRUS 40 DNA AND SPECIFIC EVENTS OF THE HOST CELL CYCLE. (E.) Pages, J. (Inst. Sci. Res. Cancer, Villejuif, France), S. Manteuil, D. Stehelin, M. Fisman, M. Marx and H. Girard. *J Virol* 12(1):99-107, 1973.

The relationship between replication of simian virus 40 (SV40) DNA and the various periods of the host-cell cycle was investigated in synchronized epithelioid CV1 cells. Cells synchronized through a double excess thymidine procedure were infected with SV40 (0.2ml) at the beginning or the middle of S, or in G₂. The first viral progeny DNA molecules were in all instances detected approximately 20 hr after release from the thymidine block, independent of the time of infection. The length of the early, prereplicative phase of the virus growth cycle therefore depended on the period of the cell cycle at which the cells were infected. Infection with SV40 was also performed on cells obtained in early G₁ through selective detachment of cells in metaphase. As long as the cells were in G₁ at the time of infection, the first viral progeny DNA molecules were detected during the S period immediately following, whereas if infection took place once the cells had entered S, no progeny DNA molecule could be detected until the S period of the next cell cycle. These results suggest that the infected cell has to pass through a critical stage situated in late G₁ or early S before SV40 DNA replication can eventually be initiated.

3873 DEOXYRIBONUCLEIC ACID OF MAREK'S DISEASE VIRUS IN VIRUS-INDUCED TUMORS. (E.) Nazeran, K. (Karolinska Inst., Stockholm, Sweden), T. Lindahl, G. Klein and L. J. Lee. *J Virol* 12(4):841-846, 1973.

RNA was extracted from [³H]thymidine-labeled Marek's disease virus (MDV) and purified by two cycles of CsCl gradient centrifugation in a fixed-angle rotor. The DNA was transcribed *in vitro* into ³²P-labeled complementary RNA (cRNA). MDV cRNA did not hybridize with DNA from chicken embryo fibroblast cultures or from chicken spleen, but hybridized efficiently with DNA from MDV particles or MDV-infected cell cultures. Marek's disease tumors from different chickens in different organs (ovary, liver, testis) were all found to contain MDV DNA sequences. The relative amount of MDV DNA varied from tumor to tumor and was between 3 and 15 virus genome equivalents/cell. The content of virus DNA/cell in spleens from tumor-bearing chickens was much lower than in tumors from the same animals. MDV-infected cell cultures con-

tained a large proportion (28-59%) of virus antigen-positive cells, as measured by immunofluorescence, but tumor cells were negative in this respect (<0.02% positive cells). These data indicate that MDV is present in tumor cells as a provirus, presumably in the form of uncoated DNA.

3874 PENETRATION OF HOST CELL MEMBRANES BY ADENOVIRUS 2. (E.) Brown, D. T. (U. Maryland Sch. Med., Baltimore) and B. T. Burlingham. *J Virol* 12(2):386-396, 1973.

Highly purified human adenovirus type 2 directly penetrated the plasma membranes of KB cells. The process of membrane penetration resulted in the appearance of large numbers of adenovirions free in the cytoplasm of the infected cells. The virions underwent a morphological change as they penetrated the cell surface. Most virions seen, by freeze-etching, in the cytoplasm and on interior membrane surfaces were distinctly rounded. Penetration of the plasma membranes and the accompanying alteration in virion morphology were dependent on a function associated with the intact cells, because neither event occurred when purified virions were added to isolated cell membranes. Inactivation of the adenovirions with heat (45 C) or antibodies before inoculation of the cells reduced the infectivity of the virus population and prevented the appearance of free virions in the cytoplasm. This concurrent reduction in plaque-forming ability and numbers of free cytoplasmic virions suggests that direct penetration of plasma vacuolar membranes is an essential step in the successful infection of host cells. The inactivation of the virions did not significantly reduce the number of virus particles found in cell vacuoles and pinocytotic vesicles.

3875 PRODUCTION OF VIRAL mRNA IN ADENOVIRUS-TRANSFORMED CELLS BY THE POST-TRANSCRIPTIONAL PROCESSING OF HETEROGENEOUS NUCLEAR RNA CONTAINING VIRAL AND CELL SEQUENCES. (E.) Wall, R. (Dept. Biol. Sci., Columbia U., New York, N.Y.), J. Weber, Z. Gage and J. E. Darnell. *J Virol* 11(6):953-960, 1973.

Adenovirus 2-transformed rat embryo cells contain virus-specific sequences which are covalently linked to cell-specific RNA sequences in heterogeneous nuclear RNA (HnRNA) molecules larger than 45S. Virus sequences are identified by hybridization to viral DNA, and the cell sequences are detected by hybridization to cellular DNA under conditions where hybridization occurs only to reiterated sites in cell DNA. Such large composite viral-cell HnRNA molecules presumably arise through the uninterrupted transcription of host sequences and integrated viral DNA. Adenovirus-specific polysomal RNA from these cells sediments as three discrete species at 16, 20, and 26S. These specific classes of viral mRNA do not contain rapidly hybridizing host-specific RNA sequences. Both virus-specific HnRNA and mRNA contain polyadenylic acid sequences since they bind to polyU columns at levels characteristic of other polyA-terminated HnRNA and mRNA. Thus, the discrete species of virus-specific mRNA in adenovirus 2-transformed cells appear to be derived from high-molecular-wt virus-specific HnRNA through a series of post-transcriptional modifications

involving polyA addition. Subsequently the HnRNA is cleaved so that the cell-specific RNA sequences that originate from the reiterated sites in cell DNA do not accompany the adenovirus mRNA to the cytoplasm. These events for the adenovirus-specific mRNA appear, therefore, to be similar to the stages in the biogenesis of the majority of mRNA in eukaryotic cells.

- 3876 ASSOCIATION BETWEEN MAREK'S HERPESVIRUS AND HUMAN CANCER. II. DETECTION OF STRUCTURAL VIRAL ANTIGENS IN CHICKEN TUMORS AND HUMAN TUMORS. (E.) Makari, J. G. (Makari Res. Lab., Englewood, N.J.). *Oncology* 28(2):177-183, 1973.

Evidence is presented which supports the previously reported findings that glycoprotein antigens from Marek's tumors have common antigenicity with those from human cancer. These glycoprotein antigens from Marek's tumors behaved in a similar manner to glycoprotein antigens obtained from Marek's herpesvirus grown in tissue culture in their cross-reactivity with the glycoprotein antigens of human cancer. For both tumor and virus antigens, the greatest degree of cross-reactivity was observed with the lymphoma-leukemia group followed by sarcomas and carcinomas. The basis for this common antigenicity is the presence of antigenic determinants for Marek's herpesvirus genome in a variety of human neoplasia. The DNA Marek's herpesvirus is thus implicated in human cancer, especially the leukemia-lymphoma and Hodgkin's group of neoplastic diseases.

- 3877 ESP-1 TYPE-C VIRUS: HELPER ACTIVITY FOR THE EXPRESSION OF LEUKEMIA IN MICE AND CHARACTERIZATION OF THE VIRUS ENVELOPE. (E.) Eckner, R. J. (Roswell Park Mem. Inst., Buffalo, N.Y.), E. S. Priori, E. A. Mirand and L. Dmochowski. *Proc Am Assoc Cancer Res* 14(March):53, 1973.

- 3878 RESPONSE OF X/Gf MICE TO ONCOGENIC VIRUSES. (E.) Goldfeder, A. (Dept. Hlth., Hosp., New York U., N.Y.). *Proc Am Assoc Cancer Res* 14(March):56, 1973.

- 3879 DETECTION OF FOAMY VIRUS IN NORMAL LACTATING MONKEY MAMMARY TISSUES. (E.) Schidlovsky, G. (Pfizer, Inc., Maywood, N.J.) and M. Ahmed. *Proc Am Assoc Cancer Res* 14(March):60, 1973.

- 3880 ENHANCEMENT OF VIRAL TRANSFORMATION OF SYRIAN HAMSTER CELLS: TEMPORAL RELATIONSHIP BETWEEN VIRUS INOCULATION AND CHEMICAL TREATMENT. (E.) Casto, B. C. (BioLabs, Inc., Northbrook, Ill.), W. J. Pieczynski and J. A. DiPaolo. *Proc Am Assoc Cancer Res* 14(March):65, 1973.

- 3881 FREEZE ETCH STUDIES OF THE SURFACES OF ONCORNAVIRUSES. (E.) Sheffield, J. B. (Inst. Med. Res., Camden, N.J.). *Proc Am Assoc Cancer Res* 14(March):71, 1973.

- 3882 INDUCTION OF LEUKEMIA, LYMPHOMA AND OSTEOSARCOMA IN THE SYRIAN GOLDEN HAMSTER BY THE ONCOGENIC DNA VIRUS SV40. (E.) Diamandopoulos, G. T. (Harvard Med. Sch., Boston, Mass.). *Proc Am Assoc Cancer Res* 14(March):100, 1973.

- 3883 DIFFERENTIAL SUSCEPTIBILITY TO TRANSFORMATION OF GENETICALLY DEFICIENT HUMAN SKIN FIBROBLASTS BY KIRSTEN-MSV AND FELINE SARCOMA VIRUS. (E.) Chang, S. S. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 14(March):103, 1973.

- 3884 TSTA OF MURINE NEOPLASMS INDUCED BY VISNA AND PROGRESSIVE PNEUMONIA VIRUSES. (E.) Law, L. W. (Natl. Cancer Inst., Bethesda, Md.) and K. K. Takemoto. *Proc Am Assoc Cancer Res* 14(March):105, 1973.

- 3885 MECHANISM OF LEUKEMOGENESIS WITH ABELSON VIRUS. (E.) Siegler, R. (Children's Cancer Res. Fdn., Boston, Mass.), H. Lazarus, I. Lane and S. Moran. *Proc Am Assoc Cancer Res* 14(March):105, 1973.

- 3886 HUMAN LEUKEMIC DNA POLYMERASES. COMPARISON OF ITS TEMPLATE-PRIMER CHARACTERISTICS WITH DNA POLYMERASES FROM HUMAN NORMAL BLOOD LYMPHOCYTES AND RNA TUMOR VIRUSES. (E.) Sarin, P. S. (Natl. Cancer Inst., Bethesda, Md.), M. C. Sarngadharan, J. Bhattacharyya and R. C. Gallo. *Proc Am Assoc Cancer Res* 14(March):107, 1973.

- 3887 AUGMENTATION OF AND INTERFERENCE WITH SPLEEN FOCI OF RAUSCHER VIRUS IN MICE INOCULATED WITH CULTURED HUMAN NEOPLASTIC CELLS. (E.) Gonzalez, F. (U. Texas M. D. Anderson Hosp., Tumor Inst., Houston), K. R. Molloy, F. Györkey and J. G. Sinkovics. *Proc Am Assoc Cancer Res* 14(March):118, 1973.

- 3888 SUBCELLULAR DISTRIBUTION OF A MURINE MYELOMA DNA POLYMERASE ACTIVITY FOUND IN PREPARATIONS OF INTRACISTERNAL A-TYPE PARTICLES. (E.) Bohn, E. W. (Natl. Inst. Hlth., Bethesda, Md.), K. K. Lueders and E. L. Kuff. *Proc Am Assoc Cancer Res* 14(March):120, 1973.

- 3889 RELEASE OF DNA POLYMERASE ACTIVITY AND SELECTIVE POLYPEPTIDES FROM THE CORE COMPONENT OF AVIAN MYELOBLASTOSIS VIRUS (AMV) BY EXPOSURE TO SODIUM CHLORIDE. (E.) Stromberg, K. (Natl. Cancer Inst., Bethesda, Md.) and S. H. Wilson. *Proc Am Assoc Cancer Res* 14(March):122, 1973.

- 3890 BIOCHEMICAL STUDIES OF THE VIRAL CORES OF THE MURINE LEUKEMIA-SARCOMA VIRUS (MOLONEY ISOLATE). (E.) Larsen, C. J. (St. Louis Hosp., Paris, France), A. Tavitian, R. Hamelin and M. Boiron. *Proc Am Assoc Cancer Res* 14(March):124, 1973.

- 3891 SINGLE CELL TRANSPLANTS OF GROSS VIRUS-INDUCED LYMPHOMAS. (E.) Vredevoe, D. L. (Sch. Nursing, U. California, Los Angeles) and E. F. Hays. *Proc Am Assoc Cancer Res* 14(March):30, 1973.
- 3892 EFFECT OF DIMETHYLSULFOXIDE AND HALOGENATED DEOXYURIDINES ON PRODUCTION OF MAMMARY TUMOR VIRUS IN TISSUE CULTURE. (E.) Lasfargues, E. Y. (Inst. Med. Res., Camden, N.J.) and J. C. Lasfargues. *Proc Am Assoc Cancer Res* 14(March):32, 1973.
- 3893 FUNCTIONAL CHARACTERISTICS OF A LYMPHOBLASTOID TUMOR CELL LINE DERIVED FROM A HERPESVIRUS SAIMIRI-INFECTED OWL MONKEY. (E.) Rabin, H. (Litton Bionetics, Inc., Kensington, Md.), W. C. Wallen, R. H. Neubauer and D. V. Ablashi. *Proc Am Assoc Cancer Res* 14(March):33, 1973.
- 3894 DETECTION OF MASON-PFIZER MONKEY VIRUS IN NORMAL MONKEY MAMMARY TISSUE AND EMBRYONIC CULTURES. (E.) Ahmed, M. (Pfizer Inc., Maywood, N.J.), W. Korol, G. Schidlovsky, J. Vidrine and S. Mayyasi. *Proc Am Assoc Cancer Res* 14(March):34, 1973.
- 3895 TRANSFORMATION OF NORMAL HAMSTER EMBRYO CELLS BY HERPES SIMPLEX VIRUS TYPE 1. (E.) Duff, R. (M. S. Hershey Med. Ctr., Pennsylvania State U.) and F. Rapp. *Proc Am Assoc Cancer Res* 14(March):37, 1973.
- 3896 SELECTIVE CYTOTOXICITY OF PERITONEAL LEUKOCYTES FOR NEOPLASTIC CELLS. (E.) Holtermann, O. A. (Roswell Park Mem. Inst., Buffalo, N.Y.), E. Klein and G. P. Casale. *Proc Am Assoc Cancer Res* 14(March):40, 1973.
- 3897 LOSS OF RESPONSE TO ERYTHROPOIETIN IN MICE AFTER INFECTION WITH FRIEND VIRUS. (E.) McGarry, M. P. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Proc Am Assoc Cancer Res* 14(March):41, 1973.
- 3898 TRANSFORMATION OF A RAT LIVER CELL LINE BY MURINE SARCOMA VIRUS (MSV). (E.) Ikawa, Y. (Natl. Inst. Hlth., Bethesda, Md.), A. Niwa, L. Tomatis, R. W. Baldwin, H. C. Chopra and A. F. Gazdar. *Proc Am Assoc Cancer Res* 14(March):109, 1973.
- 3899 "HELPER" INFLUENCE OF THE LDH-VIRUS IN THE PRODUCTION OF LEUKEMIA BY ATTENUATED RAUSCHER VIRUS. (E.) Riley, V. (Pacific Northwest Res. Fdn., Seattle, Wash.) and M. A. Fitzmaurice. *Proc Am Assoc Cancer Res* 14(March):112, 1973.
- 3900 IMMUNOLOGICALLY-ALTERED RESPONSE TO ASPARAGINASE IN THE MOUSE: INFLUENCE OF THE LDH-VIRUS. (E.) Spackman, D. (Pacific Northwest Res. Fdn., Seattle, Wash.) and V. Riley. *Proc Am Assoc Cancer Res* 14(March):113, 1973.
- 3901 EXCISION OF THE SV40 GENOME FROM TRANSFORMED CELL DNA. (E.) Butel, J. S. (Baylor Coll. Med., Houston, Tex) and V. A. L. Boyd. *Proc Am Assoc Cancer Res* 14(March):44, 1973.
- 3902 WIDE HOST RANGE OF MURINE SARCOMA VIRUS. (E.) Rhim, J. S. (Natl. Inst. Hlth., Bethesda, Md.), F. G. Duh, C. F. Demoise and R. J. Huebner. *Proc Am Assoc Cancer Res* 14(March):45, 1973.
- 3903 RIBONUCLEASE ACTIVITY AFFECTING DNA-DEPENDENT RNA POLYMERASE TEMPLATE SPECIFICITY IN NORMAL AND FRIEND VIRUS INFECTED MOUSE SPLEEN. (E.) Munson, B. R. (Roswell Park Mem. Inst., Springville, N.Y.) and C. L. Tober. *Proc Am Assoc Cancer Res* 14(March):47, 1973.
- 3904 CONTINUOUS PROPAGATION OF THE RADIATION LEUKEMIA VIRUS (RadLV) ON A C57BL MOUSE FIBROBLAST LINE. (E.) Lieberman, M. (Stanford U. Sch. Med., Calif.), O. Niwa, A. Decleve and H. S. Kaplan. *Proc Am Assoc Cancer Res* 14(March):47, 1973.
- 3905 SPONTANEOUS AND INDUCED RELEASE OF TYPE C RNA VIRUS FROM A CLONAL LINE OF SPONTANEOUSLY TRANSFORMED BALB/C 3T3. (E.) Lieber, M. M. (Natl. Inst. Hlth., Bethesda, Md.) and G. J. Todaro. *Proc Am Assoc Cancer Res* 14(March):51, 1973.
- 3906 SPECIFICITY OF ^{51}Cr -RELEASE CYTOTOXICITY BY LYMPHOCYTES IMMUNE TO MURINE SARCOMA VIRUS (MSV). (E.) Lavrin, D. H. (Natl. Cancer Inst., Bethesda, Md.), N. Soares and R. B. Herberman. *Proc Am Assoc Cancer Res* 14(March):51, 1973.
- 3907 MYCOPLASMAS PROVIDE HELPER ACTIVITY FOR FRIEND SPLEEN FOCUS-FORMING VIRUS IN MICE. (E.) Steeves, R. A. (Einstein Coll. Med., New York, N.Y.) and J. Minowada. *Proc Am Assoc Cancer Res* 14(March):71, 1973.
- 3908 PERSISTENCE OF EPSTEIN-BARR VIRUS IN EB NEGATIVE HYBRID CELLS. (E.) Glaser, R. (M. S. Hershey Med. Ctr., Pennsylvania State U.) and M. Nonoyama. *Proc Am Assoc Cancer Res* 14(March):73, 1973.
- 3909 PREVENTION OF VIRUS-INDUCED LEUKEMIA IN OFFSPRING BY MATERNAL IMMUNIZATION WITH VIRUS. (E.) Buffett, R. F. (Roswell Park Mem. Inst., Buffalo, N.Y.) *Proc Am Assoc Cancer Res* 14(March):75, 1973.
- 3910 REDUCTION OF MOUSE MAMMARY TUMOR GROWTH RATE BY STREPTOVARICIN. (E.) Kramarsky, B. (Inst. Med. Res., Camden, N.J.) *Proc Am Assoc Cancer Res* 14(March):80, 1973.

- 3911 ELECTRON MICROSCOPIC EVIDENCE TO SUPPORT THE MORPHOLOGY OF THE SEQUENTIAL FORMS IN THE ENTIRE LIFE CYCLE OF THE MURINE MAMMARY TUMOR VIRUS; A COMPARISON WITH SIMILAR FORMS IN A HUMAN MAMMARY CARCINOMA. (E.) Dolowy, W. C. (U. Washington Sch. Med., Seattle). *Proc Am Assoc Cancer Res* 14(March):2, 1973.
- 3912 DEPLETION OF SERUM GROWTH FACTORS BY 3T3 MOUSE FIBROBLASTS AND VIRAL TRANSFORMANTS. (E.) Roehm, C. J. (Pennsylvania St. Coll. Med., Hershey) and A. Lipton. *Proc Am Assoc Cancer Res* 14(March):3, 1973.
- 3913 A HIGH MAMMARY TUMOR VIRUS PRODUCING MOUSE CELL LINE. (E.) Yagi, M. J. (Dept. Bacteriology, Immunology, U. California, Berkeley). *Proc Am Assoc Cancer Res* 14(March):10, 1973.
- 3914 GROWTH OF ERYTHROBLASTOSIS VIRUS (EbV) IN TISSUE CULTURE. (E.) Darcel, C. le Q. (Animal Dis. Res. Inst., Lethbridge, Alberta, Canada). *Proc Am Assoc Cancer Res* 14(March):20, 1973.
- 3915 THE EFFECTS OF STREPTOVARICIN ON FUNCTIONS OF RNA TUMOR VIRUSES. (E.) Byrd, D. (Roswell Park Mem. Inst., Buffalo, N.Y.), E. C. Borden, J. S. Horoszewicz, L. Pothier and W. A. Carter. *Proc Am Assoc Cancer Res* 14(March):25, 1973.
- 3916 STUDIES ON THE RNA OF MASON-PFIZER MONKEY VIRUS. (E.) Harewood, K. (Pfizer Inc., Maywood, N.J.), J. Wolff, III, M. Ahmed and S. Mayyasi. *Proc Am Assoc Cancer Res* 14(March):27, 1973.
- 3917 INHIBITION OF GROSS LEUKEMIA VIRUS REPLICATION BY 3-DEAZAURIDINE. (E.) Shannon, W. M. (Southern Res. Inst., Birmingham, Ala.) and L. Westbrook. *Proc Am Assoc Cancer Res* 14(March):28, 1973.
- 3918 VIRAL INDUCTION OF HEPATIC NEOPLASMS IN LSH/LAK INBRED HAMSTERS. (E.) McCormick, K. J. (Baylor Coll. Med., Houston, Tex.), N. K. McCormick, W. A. Stenback and J. J. Trentin. *Proc Am Assoc Cancer Res* 14(March):89, 1973.
- 3919 A CELL LINE FROM A PRIMARY TUMOR ARISING IN A D2 HYPERPLASTIC NODULE. (E.) Soule, H. (Michigan Cancer Fdn., Detroit), T. Maloney, J. Vazquez and A. Long. *Proc Am Assoc Cancer Res* 14(March):90, 1973.
- 3920 PHAGOCYTOSIS OF TYPE B AND TYPE C VIRUS PARTICLES BY MOUSE PERITONEAL CELLS *IN VIVO*. (E.) Seman, G. (U. Texas M. D. Anderson Hosp., Tumor Inst., Houston) and L. Dmochowski. *Proc Am Assoc Cancer Res* 14(March):118, 1973.
- 3921 EFFECTS OF POLYADENYLIC ACIDS ON TRANSFORMATION AND ACTIVATION OF MOUSE RNA TUMOR VIRUSES. (E.) Tennant, R. W. (Biol. Division, Oak Ridge Natl. Lab., Tenn.), J. G. Farrelly and F. T. Kenney. *Proc Am Assoc Cancer Res* 14(March):125, 1973.
- 3922 SPONTANEOUS AND INDUCED NEOPLASTIC POTENTIAL OF EMBRYONAL CELL CULTURES. (E.) Yerganian, G. (Children's Cancer Res. Fdn., Boston, Mass.), A. E. Freeman and H. J. Gagnon. *Proc Am Assoc Cancer Res* 14(March):126, 1973.
- 3923 INTERRELATIONSHIPS OF CELL SHAPE, GLUCOSE TRANSPORT, AND TUMOR DEVELOPMENT IN AVIAN SYSTEMS. (E.) Perdue, J. F. (McArdle Lab., Cancer Res., U. Wisconsin, Madison). *Proc Am Assoc Cancer Res* 14(March):128, 1973.
- 3924 REPLICATION OF TYPE C VIRUS PARTICLES IN TRANSFORMED HUMAN CELLS. (E.) Maruyama, K. (U. Texas, M.D. Anderson Hosp., Tumor Inst., Houston), S. H. Wagner, G. T. O'Connor, Jr., J. L. East, S. Hiraki and L. Dmochowski. *Proc Am Assoc Cancer Res* 14(March):115, 1973.
- 3925 AN ENZYMATIC FUNCTION ASSOCIATED WITH TRANSFORMATION OF FIBROBLASTS BY ONCOGENIC VIRUSES. (E.) Unkeless, J. (Rockefeller U., New York, N.Y.), A. Tobia, L. Ossowski, J. Quigley, D. Rifkin and E. Reich. *Proc Am Assoc Cancer Res* 14(March):107, 1973.
- 3926 EFFECTS OF INHIBITORS OF PROTEIN AND NUCLEIC ACID SYNTHESIS ON THE EXPRESSION OF H-2 AND MOLONEY LEUKEMIA VIRUS-DETERMINED CELL-SURFACE ANTIGENS ON CULTURED MURINE LYMPHOMA CELLS. (E.) Cikes, M. (Karolinska Inst., Stockholm, Sweden) and G. Klein. *J Nat Cancer Inst* 48(2):509-522, 1972.

See also:

- * (Rev): 3602, 3603, 3609, 3610, 3612, 3629
- * (Chem): 3657, 3730
- * (Phys): 3782
- * (Immun): 3931, 3933, 3935, 3938, 3940, 3946, 3947, 3969, 3970, 3985, 3990, 3991, 4008, 4009, 4012, 4026, 4027, 4028, 4038, 4040, 4045, 4046

- 3927 ROLE OF CYCLIC AMP IN MITOGEN INDUCED TRANSFORMATION OF HUMAN PERIPHERAL LEUKOCYTES. (E.) Krishnaraj, R. (All India Inst. Med. Sci., New Delhi) and G. P. Talwar. *J Immunol* 111(4):1010-1017, 1973.

The effect of mitogens on adenyl cyclase activity and intracellular cAMP levels was studied in stimulated human peripheral lymphocyte cultures. Phytohemagglutinin (PHA) stimulated adenyl cyclase activity to 186% of control within 15 min following the onset of incubation. The effect was dose-dependent, with excessive concentrations of PHA causing inhibition, thus correlating with the observed pattern of PHA-induced mitogenic activity. Another mitogen, concanavalin A (Con A), was a weak stimulator of adenyl cyclase. Both PHA and Con A increased intracellular cAMP levels by 30-80%. Propranolol, chlorpromazine, and imidazole, which interfere with PHA-induced activation of adenyl cyclase or lower intracellular cAMP levels, inhibited blast transformation as measured by the rate of ³H-thymidine incorporation into DNA. Addition of cAMP to the medium produced only a two- to three-fold lymphocyte stimulation compared with the 450-fold stimulation seen with PHA. Dibutyryl cAMP, cGMP, and cCMP failed to induce transformation. It was thus concluded that the early rise in intracellular cAMP is only a part of the mechanism of PHA-induced mitogenesis.

- 3928 BCG IMMUNOTHERAPY OF LOCAL SUBCUTANEOUS GROWTHS AND POST-SURGICAL PULMONARY METASTASES OF A TRANSPLANTED RAT EPITHELIOMA OF SPONTANEOUS ORIGIN. (E.) Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England) and M. V. Pimm. *Int J Cancer* 12(2):420-427, 1973.

The potential value of bacillus Calmette-Guerin (BCG) immunotherapy in the treatment of pulmonary metastases was evaluated using a weakly immunogenic transplanted Wistar rat epithelioma. Subcutaneous injection of tumor cells (10⁵) in admixture with viable BCG (0.2-3.0 mg moist wt) retarded tumor development at this site in syngeneic rats and reduced the development of pulmonary metastases. Partial control of metastatic disease following surgical removal of s.c. tumor was obtained by i.v. injection of BCG (3.0 mg moist wt), the main response being an increase in survival (mean 75.8 days) and reduction in the number of pulmonary metastases (mean 18). Treatment was not rendered more effective by concomitant immunostimulation with irradiated tumor cells in admixture with BCG. These studies demonstrate the potential of BCG adjuvant therapy but support the concept that localization of BCG at the tumor site is necessary for effective tumor suppression.

- 3929 LYMPHOCYTE ANERGY IN PATIENTS WITH CARCINOMA. (E.) Nind, A. P. P. (Monash U. Med. Sch., Melbourne, Australia), R. C. Nairn, J. M. Rolland, E. P. G. Guli and E. S. R. Hughes. *Br J Cancer* 28(2):108-117, 1973.

None of ten patients with colonic carcinoma and none of five with malignant melanoma of skin showed

signs of immunoreactivity against cultured tumor cells by the lymphocyte populations residing within the tumors. More than half of these patients did show cytotoxic reactivity by their blood lymphocytes. Possible cytotoxic reactivity by the regional lymph node lymphocytes was also investigated in 57 tumor cases (44 colonic, 13 melanoma, and including 12 of the 15 examined for intrinsic lymphocyte activity). One-third of the cases showed positive blood lymphocyte immunoreactivity, but in only four tumors (three colonic) did the node lymphocytes show any cytotoxicity against the tumor cells. This state of anergy of intrinsic and regional lymphocytes is presumably acquired during the development of the cancer and would permit local tumor spread and metastasis to lymph nodes. Its cause has not been identified but appears to be lymphocyte inhibition rather than selective change in lymphocyte population. In particular, no special pattern is seen in the relative proportions of T and B cells in patients' blood, lymph node, or intrinsic carcinoma lymphocytes.

- 3930 IMMUNOLOGICAL ASPECTS OF RESISTANCE TO THE ONCOGENIC EFFECT OF 3-METHYLCHOLANTHRENE IN MICE. (E.) Stutman O. (U. Minnesota Med. Sch., Minneapolis). *Isr J Med Sci* 9(3):217-228, 1973.

The immunosuppressive effect of 3-methylcholanthrene (MC) was studied in relation to its role as a carcinogen using a mouse strain (C3Hf) which is sensitive and one (Strain I) which is relatively resistant to the carcinogenic action of MC. The main observations made were the following: a) Resistance to MC in I mice is not dependent on carcinogen dose. b) Inheritance of resistance in hybrids and crosses of sensitive and resistant strains is multi-factorial, with intermediate values in the F₁ hybrids. c) It is possible to predict tumor risk in F₁ hybrids by the occurrence or absence of the immunosuppressive effect of MC; the subgroups which were immunosuppressed by MC behaved as high tumor risk populations. d) The predictive value of normal versus poor immunological responsiveness to low and high tumor risk respectively could be assessed by certain tests (rejection of skin grafts across very weak histocompatibility barriers and serum agglutinin titers to brucella) but not by others (skin rejection across stronger histocompatibility barriers and serum antibody titers to sheep red cells), and could here be seen clearly in F₁ but not in F₂ hybrid populations. e) With certain MC doses in C3Hf mice, oncogenic action could be dissociated from the immunosuppressive effect. f) In the sensitive C3Hf mice there was a strict correlation between the MC dose and the degree of immunosuppression: At low doses MC did not suppress the response to sheep red cells; at intermediate dosages only the direct-hemolysin plaque-forming cells in the spleen were depressed; and at higher dosages (1.0 mg or more of MC) the indirect-hemolysin plaque-forming cells as well as the agglutinin and hemolysin titers in serum were depressed. It is inferred that immunosuppressive effect of MC may not always be a necessary component of its carcinogenic action in mice.

- 3931 NASOPHARYNGEAL CARCINOMA AND EPSTEIN-BARR VIRUS. I. FACTORS RELATED TO THE ANTI-VCA ANTIBODY. (E.) Lynn, T.-C. (Inst. Med. Sci., U. Tokyo, Japan), W.-M. Tu, T. Hirayama and A. Kawamura, Jr. *Jap J Exp Med* 43(2):121-133, 1973.

The antibody against viral capsid antigen (VCA) of Epstein-Barr (EB) virus was studied in 433 sera from 305 patients with anaplastic carcinoma of the nasopharynx in Taiwan, using the indirect fluorescent antibody method. Another 134 sera from normal people and from patients with other diseases served as controls. Patients with nasopharyngeal carcinoma had a geometric mean titer of 1:302, of which 202 sera (48.3%) were positive (titer \geq 1:640). The controls had a geometric mean titer of 1:47 and only 3 sera (2.2%) were positive. The antibody titer in 103 patients before treatment was analyzed in detail. Their geometric mean titer was 1:394, and 65 sera (63.1%) were positive. Higher anti-VCA titer was significantly associated with the development of neck lymph node metastasis. Prolonged duration of the disease without treatment also resulted in some elevation of the antibody titer. The age and sex of patients and the size of primary tumor had no significant influence on the antibody titer. There are two possible roles for EB virus in nasopharyngeal carcinoma: 1) as an oncogenic or co-carcinogenic agent or 2) as a passenger virus. Either possibility is reasonable in accordance with the fact that nasopharyngeal carcinoma, Burkitt lymphoma and infectious mononucleosis have the common feature of lymphoid tissue involvement and high anti-VCA titer.

- 3932 IgM-PRODUCING MALIGNANT LYMPHOMAS WITHOUT MACROGLOBULINEMIA. MORPHOLOGICAL AND IMMUNOCHEMICAL FINDINGS. (E.) Kaiserling, E. (Path. Inst., U. Kiel, Germany), H. Stein and K. Lennert. *Virchows Arch [Zellpathol]* 14(1):1-18, 1973.

Two malignant lymphomas were investigated. They were light microscopically characterized by a large number of PAS-positive cytoplasmic inclusions. Electron microscopically these inclusions were represented by electron-dense condensates lying in distended cisternae of the rough endoplasmatic reticulum. Immunochemical analyses revealed a high increase of IgM in the tumor tissue homogenates, but no macroglobulinemia in the sera. This indicates that the tumor cells produced but could not secrete IgM and that the cytoplasmic inclusions represented accumulated IgM. The first lymphoma showed a wide cytological spectrum from small lymphocytoid cells to cells rich in ergastoplasm. About 8-10% of these tumor cells contained cytoplasmic inclusions. In the second case the tumor consisted of mostly large cells with large nuclei and a poorly developed endoplasmatic reticulum but numerous polysomes. IgM-condensates were found exclusively in distended cisternae of the perinuclear space. These tumor cells were similar to immunoblasts in both the cytological features and site of Ig-production. In Case 1 there were no morphological clues for the cause of the secretory defect. IgM extracted from the tumor was electrophoretically identified as

mainly 8 S IgM-monomer. It is assumed that the lack of secretion was caused by a defect in the polymerization mechanism. For Case 2 it is concluded that the lack of secretion was caused by a disturbance of the IgM-transport from the perinuclear space to the Golgi apparatus due to the nearly complete absence of endoplasmatic reticulum. The two tumors represent a previously not well known group of B-cell lymphomas consisting of secretory cells which accumulate but do not secrete immunoglobulins.

- 3933 NASOPHARYNGEAL CARCINOMA AND EPSTEIN-BARR VIRUS. II. CLINICAL COURSE AND THE ANTI-VCA ANTIBODY. (E.) Lynn, T.-C. (Inst. Med. Sci., U. Tokyo, Japan), S.-M. Tu, T. Hirayama and A. Kawamura, Jr. *Jap J Exp Med* 43(2):135-144, 1973.

A total of 433 samples of serum from 305 patients, pathologically proved to be suffering from anaplastic carcinoma of the nasopharynx, were collected for clinical and immunological studies. Titration of the antibody against viral capsid antigen (VCA) of Epstein-Barr (EB) virus was done using the indirect fluorescent antibody technique with the following results: 103 patients before treatment had a geometric mean titer of 1:394, 55 patients during irradiation had a mean titer of 1:316, 60 patients at the end of radiotherapy had a mean titer of 1:327. There are no significant differences in titers of these three groups of patients. In case of recurrence, the titer was almost at the same level as that of above-mentioned groups. After remission of the disease, the antibody titer decreased in 160 patients to a geometric mean of 1:213. This is significantly low as compared with that of patients before treatment or even with that of all the patients. The further lowering of the mean titer to the level of 1:112 in 31 patients in remission for more than three yr is considered as the sequel of a decrease in antibody as well as that of the serological scar of nasopharyngeal carcinoma. Thus the anti-VCA titer can be applied clinically as a useful parameter for the diagnosis, prognosis and follow up study of the patients, and furthermore, for the detection of latent nasopharyngeal carcinoma.

- 3934 *IN VIVO* STUDIES OF CELL-MEDIATED AND HUMORAL IMMUNE RESPONSES TO ADENOVIRUS 12-INDUCED TUMOUR CELLS. (E.) Rees, R. C. (Department Med. Microbiol., U. Sheffield, England) and C. W. Potter. *Arch Gesamte Virusforsch* 4(1-2): 116-126, 1973.

CBA mice immunized with 3 or 5 doses of cell-free extract from adenovirus 12-induced tumor cells were relatively immune to subsequent challenge with 5×10^5 viable adenovirus 12-induced tumor cells. Using spleen cell transfer experiments, this immunity was shown to be cell mediated. Thus, s.c. inoculation with spleen cells from mice immunized with tumor extract together with tumor cells in a 15:1 ratio, resp., inhibited tumor growth, as shown by a reduction in tumor incidence and the size of tumors, compared to the control mice inoculated with

tumor cells and normal mouse spleen cells. When the tumor cells and spleen cells were given by different routes of inoculation, inhibition was not as marked as when the two were given together. Mice inoculated with spleen cells, from mice immune to challenge with live tumor cells (tumor immune mice) or spleen cells from mice bearing large transplantable tumors, were shown to be relatively immune to transplanted tumor cells. Transfer of serum from mice immunized with tumor extract to normal mice did not confer any immunity to transplanted tumor cell challenge; tumor growth was neither retarded nor enhanced, compared to control mice given normal CBA mouse serum. Transfer of serum from mice immune to tumor cell challenge also did not affect the growth of tumors. However, transfer of serum from tumor-bearing mice resulted in an apparent earlier development of tumors in two distinct experiments; however, in neither experiment was the difference statistically significant compared to the tumor incidence in control mice. Failure of the mice to reject a developing adenovirus 12-induced tumor may be due to two factors. By the time sensitized lymphocytes normally appear in response to a developing tumor, the tumor may have developed beyond the size at which cell-mediated immune mechanisms can reject it. Alternatively, or in addition, factors present in the serum of tumor-bearing mice may enhance tumor growth by blocking cell-mediated immune mechanisms.

3935 INDUCTION OF EPSTEIN-BARR VIRUS-RELATED MEMBRANE ANTIGENS BY 5-IODODEOXYURIDINE IN NON-PRODUCER HUMAN LYMPHOBLASTOID CELLS. (E.) Sugawara, K. (Hokkaido U. Sch. Med., Sapporo, Japan), F. Mizuno and T. Osato. *Nature [New Biol]* 246(151):70-72, 1973.

Induction of membrane-associated (MA) Epstein-Barr virus (EBV)-related antigen by 5-iododeoxyuridine (IUdR) treatment was studied *in vitro* using nonproducer NC-37 and Raji cells and producer P3HR-1 cells. Serum for indirect immunofluorescence studies was obtained from a patient with nasopharyngeal carcinoma and contained a high titer antibody to MA. Cultivation in medium containing 50 µg/ml IUdR for up to nine days resulted in an increase in MA-positive cells both producer P3HR-1 (40-70%) and nonproducer Raji and NC-37 clones (35-50%). MA induction occurred rapidly and was followed in sequence by early antigen and viral capsid antigen induction. The fraction of MA-positive nonproducer cells increased from 35% after five days of exposure to 50% when these cells were removed from IUdR and grown in regular medium. MA induction was the most marked among the three EBV-related antigens.

3936 ENHANCED TUMOR PROLIFERATION IN MICE TREATED WITH PHYTOHAEMAGGLUTININ. (E.) Pappas, A. (Med. U. Clinic, Homburg/Saar, Germany), G. Schwarze, C. Wolff and P. G. Scheurlen. *Z Immun-Infektionsforsch* 145(5):449-459, 1973.

The influence of phytohemagglutinin (PHA) on the growth of Ehrlich-ascites tumor in mice was observed

under various experimental conditions. The mitogen was given (a) before, together with, or after tumor inoculation and (b) in a single injection of 20 mg/kg body wt or in four injections of 5 mg/kg body wt at daily intervals. Challenge with a single injection resulted in an extensive and highly significant increase in tumor proliferation as compared to control animals. The same dose divided in four injections was also effective, but to a lesser extent: enhancement in tumor growth was only significant in mice receiving PHA together with or after tumor implantation. Immunosuppressive effects of PHA have been demonstrated in several animal systems. On this basis the potential of PHA as an immunosuppressive agent seems to be the most likely explanation for the increased proliferation of Ehrlich ascites tumor cells in mice. Though *in vitro* criteria are not strictly comparable with *in vivo* conditions these observations make it unlikely that the enhanced tumor proliferation observed *in vivo* under the influence of PHA may be due to a mitogenic effect of PHA on Ehrlich ascites tumor cells.

3937 IMMUNOLOGIC AND PHYSICAL CHARACTERIZATION OF HUMAN CHORIONIC GONADOTROPIN (hCG) SECRETED BY TUMORS. (E.) Vaitukaitis, J. L. (Natl. Inst. Child Hlth. Human Development, Bethesda, Md.). *J Clin Endocrinol Metab* 37(4):505-514, 1973.

Plasma, urine and tumor extracts of patients with human chorionic gonadotropin (hCG)-secreting tumors were examined for the presence of altered forms of hCG and its subunits by chromatography on a calibrated Sephadex G-100 column. Each eluate was radioimmunoassayed in homologous hCG, hCG α and hCG β assays. All patients' tumors secreted intact hCG as the predominant form of that hormone. In addition, all patients had at least one of the hCG subunits readily detectable in urine and proportionally smaller amounts in plasma, possibly reflecting differences among the plasma half-lives of hCG and its subunits. Since subunits and other aberrant forms of hCG were present in both urine and plasma, the altered forms of hCG were not an artifact of excretion. Extracts of tumor tissue strongly suggested that altered forms of hCG arise during synthesis. Qualitative and quantitative differences of hCG and its subunits extracted from the primary tumor and its metastases suggest that *in vivo* cloning of tumor cells may occur.

3938 CELL MEDIATED IMMUNITY TO HERPESVIRUS TYPE 1 IN CARCINOMA AND PRE-CANCEROUS LESIONS. (E.) Lehner, T. (Guy's Hosp. Med. Dental Sch., London, England), E. J. Shillitoe, J. M. A. Wilton and L. Ivanyi. *Br J Cancer* 28(Suppl. 1):128-134, 1973.

The response of lymphocytes to *Herpesvirus hominis* type 1 (HVH1), *Candida albicans* and phytohemagglutinin was studied sequentially over a period of three yr in patients with leukoplakia and carcinoma. In the keratosis-acanthosis group of leukoplakia there was a significant decrease in stimulation of lymphocytes by HVH1, in contrast to epithelial atypia which yielded both increased stimulation indices and macro-

phage migration inhibition to HVH1. Non-specific depressed cell mediated immune responses were found in carcinoma. Sequential data revealed major fluctuations in stimulation indices to HVH1 during the course of epithelial atypia and a fall in the stimulation indices from > 7 to < 2 was associated with carcinomatous transformation. These changes argue in favor of participation of HVH1 in the pathogenesis of some leukoplakias, and the development of epithelial atypia with subsequent carcinoma might be a function of the cell mediated immune responses to the virus.

3939 THE ROLE OF CIRCULATING ANTIGEN AS AN INHIBITOR OF TUMOUR IMMUNITY IN MAN. (E.)

Currie, G. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England). *Br J Cancer* 28(Suppl. 1): 153-161, 1973.

The cytotoxic effects of lymphocytes on autologous tumor cells in patients with a variety of diseases including malignant melanoma, bladder carcinoma, hypernephroma and sarcomas were examined. A serum inhibitor of cell-mediated immunity was indentified in these patients. The inhibitor is specific to tumor type, its presence correlates with clinical status, and it has an apparent affinity for the lymphocyte surface from which it can be removed by extensive washing. The most likely candidate for such material seemed to be tumor associated antigen shed from the cells of the tumor. This hypothesis was tested by fractionating the inhibitory sera of two cancer patients on a molecular wt basis. In one case, the inhibitory activity was exclusively in the 30-100,000 dalton range, indicating that the serum inhibitor cannot be immunoglobulin and is presumably free antigen with or without other proteins. In the other case, with regional node involvement, there was activity in the 30-300,000 dalton range, indicating that both immune complex and excess free antigen may be present.

3940 IMMUNOFLUORESCENT ANTIGEN ASSOCIATED WITH EPSTEIN-BARR VIRUS INDUCED BY 5-iodo-DEOXYURIDINE. (E.) Sugawara, K. (Hokkaido U. Sch. Med., Sapporo, Japan) and T. Osato. *Nature [New Biol]* 246(151):72-73, 1973.

The activation of Epstein-Barr virus (EBV)-associated antigens by 5-iododeoxyuridine (IUdR) was studied by indirect immunofluorescence staining in several producer and nonproducer cell lines. Sera used for testing were obtained from patients with nasopharyngeal carcinoma or Burkitt's lymphoma and from normal donors. Nonproducer as well as producer cells exposed to IUdR showed a weak but distinct nuclear fluorescence occurring as early as one day after exposure and lasting for several days. No such early nuclear fluorescence was seen in human embryo fibroblasts, cord leukocytes, or HeLa cells. This fluorescence may represent a new antigen, tentatively designated "early nuclear antigen" (ENA), distinct from the three known EBV-related immunofluorescent antigens, membrane antigen (MA) early antigen (EA), and viral capsid

antigen (VCA). ENA fluorescence was achieved exclusively by EBV-positive human sera and not negative sera. It did not represent MA or EA since it stained with EBV-positive normal sera not containing antibodies to these antigens. It was probably not VCA since VCA fluorescence is cytoplasmic.

3941 IMMUNOLOGICAL TUMOR ENHANCEMENT AND *IN VITRO* CYTOTOXICITY. FURTHER EVIDENCE FOR THE INVOLVEMENT OF 7S γ 2a-IMMUNOGLOBULIN. (E.) Eustace, J. C. (U. Miami Sch. Med., Fla.) and G. L. Irvin, III. *Transplantation* 16(3):171-176, 1973.

The ability of enhancing antiserum to facilitate EL4 tumor growth after the selective removal of 7S γ 2a- or 7S γ 1-immunoglobulin classes by absorption with specific antisera was tested. Mice receiving antiserum with 7S γ 1-immunoglobulin class removed showed enhanced tumor growth and death. Mice receiving antiserum with 7S γ 2a-globulin class removed by immunoabsorption showed no enhanced tumor growth. Antiserum preparations identical to those used for the *in vivo* experiments were tested for their ability to lyse EL4 cells *in vitro*. Antiserum with 7S γ 1-immunoglobulins removed had a complement-dependent cytotoxic titer equal to that of the unabsorbed known positive control. Antiserum with 7S γ 2a-globulin removed did not lyse EL4 cells above normal serum control levels. These results supply further evidence that the immunoglobulin class responsible for enhancing antibody activity *in vivo* and for complement dependent cytotoxic activity *in vitro* is 7S γ 2a.

3942 A MODEL FOR IMMUNITY TO MELANOMAS IN MICE. (E.) Burger, D. R. (VA Hosp., Portland, Ore.), F. Hu, L. M. Pasztor and A. Malley. *Proc Soc Exp Biol Med* 144(2):426-430, 1973.

The relationship between humoral and cellular immunity in protecting immunized C57BL/6 mice from development of melanomas following challenge with P51 mouse melanoma cells was studied. Mice were immunized with a single s.c. injection of killed P51 cells in complete Freund's adjuvant. Whereas all ten of the untreated controls and nine of ten adjuvant-treated controls challenged with 10^6 viable P51 cells developed melanomas within 14 days, none of the immunized animals so challenged had visible tumors at this time. After 20 days, however, mean tumor wts of control and immunized mice were not significantly different. Tumors wts in adjuvant-treated controls were significantly less after 20 days than those of the other groups. Regardless of treatment, all animals with tumors weighing over 3 g developed detectable tumor-specific antibody. Immunized mice with tumors weighing less than 3 g also had detectable antibody levels. Immunized mice also developed cellular immunity as determined by *in vitro* indirect migration inhibition tests. Spleen, lymph node, and peritoneal exudative cells from immunized mice passively transferred melanoma immunity to normal mice, which were resistant to tumor cell challenge two days, but not 16 days, after adoptive transfer.

- 3943 A STUDY OF HERPES SIMPLEX TYPE 2 ANTI-BODY STATUS IN GROUPS OF PATIENTS WITH CERVICAL NEOPLASIA IN CZECHOSLOVAKIA. (E.) Janda, Z. (Inst. Sera and Vaccines, Prague, Czechoslovakia), J. Kanka, V. Vonka and B. Svoboda. *Int J Cancer* 12(3):626-630, 1973.

The incidences of antibodies to herpes simplex virus (HSV) types 1 and 2 were determined in 79 females with cervical atypia or different stages of cervical cancer and in 75 age and socioeconomically matched control females with benign disease of the cervix. Virus neutralization studies using patients' sera showed that although some subjects from all groups had antibodies to HSV-1 and HSV-2, the percentage of patients with anti-HSV-2 antibodies was significantly increased in those groups with cervical atypia (50%), *in situ* carcinoma (48%), and invasive cervical carcinoma (50%). The overall incidence of anti-HSV-2 antibodies among controls was 19%. The geometric mean titers for both HSV-1 and HSV-2 antibodies were higher in the invasive carcinoma group than in any other group. The finding of increased anti-HSV-2 titers in cervical carcinoma patients of this series from Prague agrees favorably with results published in the general literature.

- 3944 ALPHAFETOPROTEIN IN TESTICULAR TUMORS. (E.) Merrin, C. (Roswell Park Memorial Inst., Buffalo, N.Y.), E. Sarcione, M. Bohne and D. J. Albert. *J Surg Res* 15(4):309-312, 1973.

The presence of α_1 -fetoprotein in the serum of 50 patients (aged 8-83 yr) with testicular tumors of embryonal origin was evaluated by the double-diffusion technique on Ouchterlony plates, immunoelectrophoresis, and crossover electrophoresis. Of the 50 patients, 36 were clinically cured and 14 had active disease. The testicular tumors comprised 27 teratocarcinomas, 18 embryonal cell carcinomas, and 5 mixed tumors. All patients with clinically inactive tumors were α_1 -fetoprotein negative. Of the 14 patients with active disease, 11 were negative and 3 were positive. Two of the positive patients had teratocarcinoma and one had embryonal cell carcinoma. During combined adriamycin and cyclophosphamide therapy the serum α_1 -fetoprotein of one teratocarcinoma patient decreased from 2.2 mg% to 0. A similar decrease (2.2 mg% to 1) was observed in the embryonal cell carcinoma patient during treatment with adriamycin plus dichloro-diamino platinum. The second patient with teratocarcinoma died before treatment could be initiated; at the time of death the α_1 -fetoprotein increased from 25.2 to 42.8 mg%.

- 3945 EVALUATION OF LEUCOCYTE FUNCTIONS SIX YEARS AFTER TUMOUR AUTOGRAFT IN HUMAN MAMMARY CANCER. (E.) Anderson, J. M. (Glasgow Royal Infirmary, Scotland), F. Kelly, S. E. Wood, K. D. Rodger and R. I. Freshney. *Br J Cancer* 28(1):83-96, 1973.

Mammary cancer directed and nonspecific immunoassays were made in three groups, each of 11-16 female patients. One group had primary mammary cancer treat-

ed by mastectomy and postoperative radiotherapy plus an autograft of irradiated tumor (AIT) 40-66 months previously. A second age-matched group had mammary cancer comparable to the first group in clinical presentation and treatment except that no AIT was given. The third group consisted of non-cancer-bearing age-matched females. The migration of leucocytes from autografted patients was significantly inhibited in the presence of allogeneic mammary cancer cells from a standardized panel, compared with leucocytes from either non-autograft patients or non-cancer bearers. Selected data from a lymphocyte cytotoxicity test revealed a significantly greater kill of allogeneic mammary cancer target cells by autograft lymphocytes than by those of other groups. These indications of increased cancer directed cell-mediated immunity in respect of sensitivity and toxicity in association with AIT require further elucidation under strictly controlled conditions.

- 3946 CHANGES IN CELL SURFACE PROPERTIES DURING THE *IN VIVO* GROWTH OF MOLONEY LYMPHOMA. (E.) Fenyö, E. M. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), P. T. Peebles, A. Wahlström, E. Klein and A. J. Cochran. *Isr J Med Sci* 9(3):239-250, 1973.

Cell surface properties of the Moloney lymphoma YAC were studied during *in vivo* ascites growth in mice. Strain A/Sn and (A.BY x DBA/2)F and (A/Sn x C3H)F hybrids were used. There was an initial increase in the percentage of large cells, cytotoxic sensitivity, electrophoretic mobility and the concentration of Moloney-specific surface antigen. The cytotoxic sensitivity of cells growing in irradiated or neonatally virus-infected hosts was high at the onset. Days 7 to 9 after i.p. inoculation of the neoplastic cells were found to be critical in the development of altered cell surface characteristics. Subsequently, when logarithmic cell growth stopped, sensitivity to immune cytolysis, Moloney-specific antigen concentration, negative surface charge, cell motility and the proportion of large cells decreased, while virus release in short-term *in vitro* cultures increased. The decrease in surface antigen concentration is probably a contributing factor in the decline of immunosensitivity. Since immunosensitivity decreased in irradiated and neonatally virus-infected mice as well, and was also apparent when isoantisera was used, host immune factors cannot be held solely responsible.

- 3947 AVIAN LEUKOSIS-SARCOMA VIRUS ANTIBODIES IN WILDFOWL, DOMESTIC CHICKENS AND MAN IN KENYA. (E.) Morgan, H. R. (U. Rochester Sch. Med. Dentistry, New York). *Proc Soc Exp Biol Med* 144(1):1-4, 1973.

The occurrence of infections with one or more subgroups of avian leukosis-sarcoma viruses (ALV) was indicated by the demonstration of neutralizing antibodies for these viruses in wildfowl and domestic chickens. These infections persist under natural

conditions in the African bush among wildfowl as well as in chickens maintained in isolated villages or a commercial poultry farm. Infections with ALV subgroups A and B were common and some evidence was obtained for infection with subgroup D but subgroup C viruses appeared to be absent. Antibodies for ALV were also found in 14 of 68 human subjects living in the areas studied. These data suggest that ALV are of ancient origin in nature and have evolved and spread with the domestication of wildfowl.

- 3948 SERUM-MEDIATED LEUKEMIA CELL DESTRUCTION IN AKR MICE. ROLE OF COMPLEMENT IN THE PHENOMENON. (E.) Kassel, R. L. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), L. J. Old, E. A. Carswell, N. C. Fiore and W. D. Hardy, Jr. *J Exp Med* 138(4):925-938, 1973.

AKR mice with spontaneous leukemia were infused i.v. with normal serum from a variety of species. Leukemia cell destruction was produced by serum from strains of mice (Swiss, C57BL/6, B10D2 new, SJL/J) possessing the full spectrum of complement components, but not by serum from strains (AKR, A, B10D2 old, DBA/2) with a genetically determined deficiency of C5. Serum from guinea pigs, horses, and humans also causes destruction of leukemia cells. The antileukemic factor in normal serum was heat labile (56 C for 35 min) and could be inactivated by cobra venom factor (CVF). Tests of individual complement factors from guinea pig serum and human serum suggest that C5 is the antileukemic complement component in normal serum. Evidence was obtained that complement also plays a role in the antileukemic effect of interferon and endotoxin. Infusion of C5 consistently reduced leukemic nodes and spleen in AKR leukemic mice, while other complement components were generally ineffective. These agents caused a remarkable reduction in leukemic lymph nodes and spleen 24 hr postadministration; in leukemic mice pretreated with CVF, a pronounced antileukemic effect was apparent by 48 hr after interferon and endotoxin infusion.

- 3949 SEROLOGIC STUDIES OF INFECTIONS IN PATIENTS WITH HEMATOLOGIC MALIGNANCY. (E.) Strangnegard, O. (Inst. Med. Microbiol., U. Gothenburg, Sweden), S. E. Holm, A. Weinfeld and J. Westin. *Scand J Inf Dis* 5(3):181-186, 1973.

Serologic studies were conducted on 77 patients with various hematologic malignancies seen at Sahlgren's Hospital, Gothenburg, Sweden, from 1971 to 1972, to determine the type and incidence of concomitant infections. Thirty two had lymphoproliferative disorder, 12 had Hodgkin's disease, 18 had myeloproliferative diseases and 15 had acute leukemia. Results were compared with those from 60 age and sex matched controls. Significantly increased frequencies of elevated antibody titers, especially against cytomegalovirus (CMV) and Epstein Barr virus (EBV) were seen. Elevated titers against other herpes simplex viruses were also measured. These infections appeared to be largely due to reactivation of infectious agents, as anti-virus antibodies were

present initially in most patients. Most (84%) of the patients showing serially increasing antibody titers were on immunosuppressive treatment, compared to only 51% of patients without serological evidence of infection. Some patients showed significant simultaneous titer increases against up to four infectious agents. Bacterial infections were encountered less frequently than were herpes group virus infections. Toxoplasmosis was observed in three patients, one of whom died of toxoplasma encephalitis.

- 3950 HETEROLOGOUS ANTILYMPHOCYTE SERUM HASTENS THE GROWTH OF 7,12-DIMETHYLBENZ(α)ANTHRA-CENE INDUCED TUMOURS IN MICE. (E.) Baroni, C. D. (Inst. Path. Anat. II, U. Rome, Italy), R. Scelsi, M. L. Peronace and S. Uccini. *Br J Cancer* 28(3):221-226, 1973.

The possible effects of repeated injections of rabbit anti-mouse lymphocyte serum (ALS) or normal rabbit serum (NRS) on 7,12-dimethylbenz(α)anthracene (DMBA) induced tumorigenesis were studied in Charles-River mice. ALS or NRS were given either at the time of DMBA administration and subsequently at weekly intervals for the first 10 wk of life, or at daily intervals for seven days during the first, second, third or fourth wk of life. Treatment of mice during the first wk of life significantly increased the number of histologically verified malignant lymphomas. Treatment for the first 10 wk decreased the mean survival time of tumor-bearing animals. A significantly increased incidence of lung tumors was observed in DMBA-treated mice which received ALS during the second wk of life. ALS treatment of any of the other groups resulted in tumor incidence not significantly different from those observed in the DMBA-and NRS-treated controls.

- 3951 TISSUE IMMUNOGLOBULINS IN NODULAR LYMPHOMAS AS COMPARED WITH REACTIVE FOLLICULAR HYPERPLASIAS. (E.) Braylan, R. C. (Dept. Path., U. Chicago Ill.) and H. Rappaport. *Blood* 42(4):579-589, 1973.

Frozen tissue sections of lymph nodes and spleens from 12 patients with nodular (follicular) lymphomas were studied, with fluorescein-labeled antisera against human immunoglobulins. Immunoglobulins detected by this method in the neoplastic nodules were minimal or absent. Furthermore, in no instance could the characteristic network pattern of immunoglobulin distribution, which is readily demonstrable in active germinal centers, be observed within these nodules. It is suggested that the preservation of the follicular network of immunoglobulins, as observed in reactive follicular hyperplasias by fluorescent antihuman IgG, IgM, or polyvalent antisera, indicates functionally intact germinal centers. The detection, by this rather simple method, of large amounts of immunoglobulin distributed in such a distinctive pattern may thus be very useful in differentiating between severe benign follicular hyperplasia and malignant lymphoma with nodular pattern. The two conditions are not easily distinguished by routine histologic methods.

3952 SIGNIFICANCE OF TUMOUR ASSOCIATED ANTIGENS ON HUMAN COLONIC CARCINOMATA. (E.)

Embleton, M. J. (Cancer Res. Campaign Lab., U. Nottingham, England). *Br J Cancer* 28(Suppl. 1):142-152, 1973.

A microcytotoxicity assay was used to detect cell mediated immunity against colon and rectum carcinoma cells *in vitro*. Lymphocytes from 29 (62%) of 47 colon carcinoma patients were cytotoxic for the cells, but no cross reactions between colon carcinomas and tumors of other types were observed. The effect of papain solubilized tumor membrane extracts was evaluated by testing lymphocytes for reactivity following preincubation with the extracts. Soluble preparations of pooled colon carcinomas inhibited cytotoxicity by sensitized lymphocytes, but similar extracts of normal colon or melanoma had no inhibitory effect. The results suggest that soluble tumor antigen may play a role in abolishing lymphocyte reactivity, and this supports the concept that cellular immunity against tumors *in vivo* may be inhibited by circulating antigen.

3953 IMMUNOLOGICAL STUDY OF CARCINOEMBRYONIC ANTIGEN (CEA) AND A RELATED GLYCOPROTEIN.

(E.) Darcy, D. A. (Chester Beatty Res. Inst. Sutton, England), C. Turberville and R. Janes. *Br J Cancer* 28(2):147-160, 1973.

A comparison was made of the immunological properties of carcinoembryonic antigen (CEA) and another perchloric acid-soluble macromolecule which occurs in colonic and certain other carcinomas and which is here termed CEX. By using a variety of antisera it was shown that the two substances share common antigenic groups as well as having characteristic ones of their own. These latter groups enabled the preparation of antisera which give a gel diffusion line only with CEA and an antiserum which gives a line only with CEX. No immunological difference could be detected between CEX and the normal glycoprotein of Mach or the nonspecific cross-reacting antigen of von Kleist and Burtin. CEX was found in fetal gut, in plasma, and associated with CEA in virtually all the tissues and fluids in which the latter occurs; the two appear to go hand-in-hand. There was no evidence that CEX is less or more cancer-specific than CEA — it is merely found in greater quantity; neither substance showed absolute cancer specificity. The usefulness of a radioimmunoassay for CEX is discussed, and also the possibility of interference by CEX in the radioimmunoassay for CEA. Evidence of two molecular species of CEA was obtained.

3954 LYMPHOCYTE BINDING OF AGGREGATED IgG AND SURFACE Ig STAINING IN CHRONIC LYMPHOCYTIC LEUKAEMIA. (E.)

Dickler, H. B. (Rockefeller U., New York, N.Y.), F. P. Siegal, Z. H. Bentwich and H. G. Kunkel. *Clin Exp Immunol* 14(1):97-106, 1973.

Eleven selected patients with chronic lymphocytic leukemia were evaluated for lymphocyte binding of

aggregated IgG and surface Ig staining in order to classify them into B and T cell types. The lymphocytes of 10 of the 11 patients bound aggregates and stained for surface Ig. In the individual ten patients the number of cells binding aggregates was high (88-100%, mean 96%) whereas the number staining for surface Ig was more variable (8-100%, mean 62%). Parallel and double labelling experiments with aggregates and sheep red blood cell rosettes, a human T cell marker, provided evidence that aggregates were binding to B cells only, even when surface Ig was not detectable. Aggregates did not bind to human thymocytes. Lymphocytes from some cases of CLL have low but not absent amounts of surface Ig that may be only partially detected by fluorescence techniques. Aggregate binding appears to be a more sensitive method for the detection of B lymphocytes than surface Ig staining. It appears that a sandwich technique enhances visualization of small aggregates which bind to lymphocytes but are not visualized by the direct method. In one of the eleven patients the leukemic cells were negative in the aggregate binding test. Separate studies on this case also indicated an absence of surface Ig staining and a high percentage of cells forming sheep red blood cell rosettes. It would appear that this case represented a T cell leukemia.

3955 STUDIES ON TUMOR-ASSOCIATED ANTIGEN: TAA.

(E.) Lo Gerfo, P. (Coll. Physicians Surgeons, Columbia U., New York, N.Y.) F. P. Herter, V. Li Volsi and S. Bennett. *J Surg Res* 15(4):290-294, 1973.

Two tumor-associated antigens with immunological identity were demonstrated in tissue from a patient with metastatic colonic adenocarcinoma. The antigens are separable by ion-exchange chromatography and electrophoresis. They appear identical by Ouchterlony immunodiffusion analysis and by Sephadex chromatography. The differences in electrophoretic mobility of these antigens appear related to a sialic acid moiety, although it was demonstrated that the antigenic specificity does not reside in this portion of the molecule. These antigens share at least one common determinant with a low-molecular-wt substance found in perchloric acid extracts of normal lung and colon. This and previous studies would suggest that this antigen is identical to carcinoembryonic antigen.

3956 CELL-SURFACE GLYCOPROTEINS OF TWO SUBLINES OF THE TA3 TUMOR. (E.)

Codington, J. F. (Harvard Med. Sch., Boston, Mass), B. H. Sanford and R. W. Jeanloz. *J Natl Cancer Inst* 51(2):585-591, 1973.

Incubation of cells with proteolytic enzymes, followed by fractionation on chromatographic columns, revealed a glycoprotein material of high molecular wt on the surface of cells of the nonstrain-specific TA3-Ha subline of the strain A mouse ascites tumor. This material was not observed in fractions obtained by similar procedures from cells of the strain-specific TA3-St subline. Although the material from TA3-Ha cells strongly inhibited agglutination of

NN-specific human erythrocytes by an extract of *Vicia graminea* seeds, no fractions of demonstrable activity were obtained by proteolysis from TA3-St cells, which suggests the absence of carbohydrate groups of similar structure in material removed from TA3-St cells. Incubation of the two sublines with neuraminidase (*Vibrio cholerae*) removed 2.3 times as much sialic acid/unit surface area from the TA3-Ha cell as from the TA3-St cell. The ratio of *N*-acetyl- to *N*-glycolylneuraminic acid from the TA3-Ha subline was approximately 13:1 and from the TA3-St about 4:1. The correlation of loss of strain specificity in the TA3-Ha cell with the presence of this unique large-surface glycoprotein suggests that this material may mask surface histocompatibility antigens.

- 3957 INTERFERENCE OF IMMUNOLOGIC SURVEILLANCE BY IMMUNOREGULATORY ALPHA GLOBULIN - A HYPOTHESIS. (E.) Ablin, R. J. (Memorial Hosp., Springfield, Ill.). *Neoplasma* 20(2):159-162, 1973.

Based upon studies demonstrating that "Immunoregulatory Alpha Globulin" (IRA) can effectively suppress antigen-induced proliferation of previously sensitized lymphocytes and suppress both the primary and secondary immunologic response, it is hypothesized that IRA may suppress, to varying degree, the host's mechanism(s) of immunologic surveillance. Data obtained from the study of the alterations of serum proteins in 23 patients with carcinoma of the prostate is presented as evidence in support of this hypothesis.

- 3958 THE MACROPHAGE AGGREGATION ASSAY FOR CELL-MEDIATED IMMUNITY IN MAN. STUDIES OF PATIENTS WITH HODGKIN'S DISEASE AND SARCOIDOSIS. (E.) Gotoff, S. P. (Michael Reese Hosp., Chicago, Ill.), S. Lolekha, M. Lopata, J. Kopp, R. L. Kopp and T. J. Malecki. *J Lab Clin Med* 82(4):682-691, 1973.

A new *in vitro* method for testing cell-mediated immunity in man was compared with delayed hypersensitivity skin tests and tritiated thymidine incorporation by peripheral blood lymphocytes in response to antigen. Aggregation of guinea pig peritoneal exudate cells was observed after incubation with supernatant fluids from cultures of sensitive human peripheral blood lymphocytes with specific antigens. *Candida albicans*, purified protein derivative (tuberculin, PPD), histoplasmin, and streptolysin O were used in the *in vitro* studies. *Candida*, PPD, histoplasmin, trichophyton, mumps, and streptokinase-streptodornase were used for delayed hypersensitivity skin tests. In normal adults tested with *candida*, PPD, and histoplasmin, the presence of macrophage aggregation factor (MAF) correlated with tritiated thymidine incorporation in 82% and with skin test results in 84% of the tests. Streptolysin O stimulated MAF synthesis in 19 of 20 normal adults and all showed an increase in DNA synthesis. In contrast, the *in vitro* and *in vivo* responses were diminished in 14 patients with Hodgkin's disease and 28 patients with sarcoidosis. Twenty-one percent of the patients with Hodgkin's disease and 50% of the patients with

sarcoidosis reacted to one or more of the skin-test antigens. MAF activity was observed in 28% of the patients with Hodgkin's disease and 32% of those patients with sarcoidosis. Lymphocytes from 64% of the Hodgkin's patients and 61% of the sarcoid patients synthesized DNA in response to antigen. While antigen-stimulated DNA synthesis by lymphocytes appears to reflect a cellular recognition phenomenon which may not be limited to cell-mediated reactions, the macrophage aggregation assay has been shown to correlate with delayed hypersensitivity in the guinea pig. Its adaptation for the study of cell-mediated immunologic functions in man provides a new approach to immunodeficiency and autoimmune diseases.

- 3959 DETECTION BY IMMUNOFLOUORESCENCE OF INTRACYTOPLASMIC ANTIGENS IN CELL LINES DERIVED FROM HUMAN SARCOMAS. (E.) Moore, M. (Robert Jones & Agnes Hunt Orthopedic Hosp., Oswestry, England), P. G. Witherow, C. H. G. Price and S. A. Clough. *Int J Cancer* 12(2):428-437, 1973.

Fourteen cell lines originating from a variety of tumors of human connective tissue including osteosarcoma (6), fibrosarcoma (3), chondrosarcoma (2), rhabdomyosarcoma (1), Ewing's tumor (1), and chordoma (1) were maintained in tissue culture for periods of 2-48 wk. During their term in culture, acetone-fixed monolayers of cells were serially subjected to indirect immunofluorescence (IF) tests using serum from cancer patients and individuals without malignancy, in an attempt to detect intracytoplasmic sarcoma-associated antigens. Antigen-positive cells were demonstrated in 10/14 cell lines, although their expression in tissue culture was variable, and cells exhibiting their IF features could not be unequivocally detected in the original tumor specimens. Such cells were present in cultures derived from different types of sarcomas, indicating the cross-reactive nature of the antigens associated with them. The overall incidence of antibodies reactive with the cell lines in sarcoma patients in various phases of disease was 13/34 (38%), but antibodies were only occasionally detected in the sera of individuals without malignancy or with histologically unrelated neoplasms. Similar antigen-positive cells could not be identified in 15 tissue cultures of comparable age *in vitro* originating from normal adult, carcinoma, or embryonic tissue or in seven long-established cell lines, thereby implying a degree of specificity for sarcoma-derived cell lines. The nature of the IF+ cells and of the antigen(s) expressed by them is discussed.

- 3960 CIRCULATING ANTIBODIES IN HUMAN MALIGNANT DISEASE. (E.) Whitehouse, J. M. A. (St. Bartholomew's Hosp., London, England). *Br J Cancer* 28(Suppl. 1):170-174, 1973.

In an attempt to categorize some of the antibodies to be found in the serum of patients with untreated malignancies, 113 sera from patients with cancer (hypernephroma, malignant melanoma, neuroblastoma, carcinoma of the bronchus, breast and ovary) and 46 sera from healthy individuals having the same age

range as the cancer patients were screened for auto-antibodies. Cryostat sections of 6 μ m thickness of rat stomach, liver and kidney were prepared. Coded sera were then examined on this substrate by indirect immunofluorescence. Any sera found to contain antibodies were titrated. Antibodies to mitochondria, renal tubules or gastric parietal cells were very infrequent. Antinuclear factor (ANF) was found in 27% of the cancer sera, being present in all the conditions except neuroblastoma. Two per cent of control sera contained ANF. Smooth muscle antibody (SMA) was found in 68% of sera from cancer patients and in 20% from the controls. In 7/21 of the neuroblastoma sera an IgG antibody was found, and SMA was found in 16/21 sera. In a subsequent experiment antimicrotubular antibody was found in infectious mononucleosis sera. It is believed to react with an antigen closely related to cytoplasmic microtubules and is capable of cross reacting widely with many different fixed cells, making the identification of tumor specific antibodies more complex. A preliminary study of sera from 25 untreated malignant melanoma patients, 15 recurrent malignant melanoma patients and 16 normal healthy subjects suggests that although there are antibodies in the sera of healthy individuals and in those with malignant melanoma which cross react, there exists in the sera of many of these patients an antibody with a greater affinity for the cytoplasm of melanoma cells than that of a non-malignant cell.

3961 HUMAN BREAST CANCER. A MODEL FOR CANCER IMMUNOLOGY. (E.) Black, M. M. (New York Med. Coll., New York). *Isr J Med Sci* 9(3):284-299, 1973.

Cellular manifestations of lymphoreticuloendothelial responses in primary breast cancers and in the draining axillary lymph nodes are reviewed and are found to correlate with stage and survival characteristics. The findings suggest that human breast cancer cells are antigenic and provoke prognostically significant immunological responses. The latter appear to involve cellular hypersensitivity rather than antibody-mediated reactions. These conclusions are supported by observations on cellular responses to sections of autologous breast cancer tissue on skin window preparations and by finding that second breast cancers arising after a primary *in situ* breast cancer are characterized by unusually favorable stage and survival.

3962 IMMUNE RESPONSIVENESS IN HODGKIN'S DISEASE. (E.) Denton, P. M. (Royal Marsden Hosp., Sutton, England). *Br J Cancer* 28(1):119-127, 1973.

Several features of immune reactivity in Hodgkin's disease are reviewed and work performed by the author is described. Immunoglobulin containing cells have been identified in varying proportions according to the histological stage of the disease. Most of the immunoglobulin was demonstrated to be of the IgG class. With regard to cell mediated immunity in Hodgkin's disease, observations on 17 untreated patients suggest that there is no impairment of lymphocyte

transformation induced by allogeneic cell stimulation *in vitro*. However, commencement of radiotherapy abolishes the reaction and its effects are prolonged. A preliminary report is presented on the antigen associated with Hodgkin's disease which was first identified by Order, Porter and Hellman in 1971. This antigen has now been found in splenic tissue from a variety of neoplasms of the lymphoreticular system.

3963 SPECIFIC LYMPHOCYTOTOXICITY AND BLOCKING FACTORS IN TUMOURS OF THE CENTRAL NERVOUS SYSTEM. (E.) Kumar, S. (Christie Hosp., Manchester, England) and G. Taylor. *Br J Cancer* 28(1):135-141, 1973.

The results of lymphocytotoxicity tests, in autologous and allogeneic combinations, on 23 human central nervous system tumors (astrocytoma, medulloblastoma, ependymoma, oligodendroglioma and a mixed glioma) are presented. The specific lymphocytotoxicity was directed against morphologically distinguishable neoplastic cells and this reactivity was "tumor type" and not "brain tumor specific". Serum samples from patients with clinically active glial tumors had activity which significantly cancelled the specific cytotoxicity of autologous lymphocytes. Under similar cultural conditions no sera from 11 healthy donors, and only one of 14 patients who had been clinically free of the disease for more than one year, possessed blocking activity. Therefore, whatever the nature of these blocking factors (antigen-antibody complex or soluble antigen), it is likely that a regular follow-up of the patient's lymphocytotoxicity and blocking activity might provide a guide to the progress of the disease.

3964 THYMUS FUNCTION IN SPONTANEOUS LYMPHOID LEUKEMIA. I. PREMATURE LEUKEMOGENESIS IN "YOUNG" THYMECTOMIZED MICE BEARING "OLD" THYMUS GRAFTS. (E.) Nagaya, H. (Duke U. Med. Ctr., Durham, N.C.). *J Immunol* 111(4):1048-1051, 1973.

More than 90% of 43 AKR mice died of spontaneous lymphoid leukemia before the age of 12 months. However, the mice did not develop leukemia until the age of 6 months suggesting that a latent period or aging was necessary for the development of leukemia. Moreover, the incidence of leukemia was reduced to nil in 24 mice thymectomized before the age of 6 months. In a study of the thymic influence on leukemogenesis, 1-month-old AKR mice were thymectomized and received syngeneic 5- to 6- or 1-month-old thymus grafts. Of the 21 mice so treated, 13 (62%) died of leukemia before the age of 6 months. Only 3 of 17 control mice thymectomized at the age of 1 month and grafted with 5- to 6-month-old spleens died of leukemia. When 5- to 6-month-old mice were thymectomized and grafted with thymus glands from 1-month-old mice, none of 19 mice developed leukemia for at least 5 months or until the recipient mice were 11 months old. The latent period for leukemogenesis appears to be required by the reticular epithelial cells of the thymus grafts rather than by the lymphoid cells of host origin migrating from bone marrow to thymus.

- 3965 TRANSPLANTABLE IMMUNOGLOBULIN-SECRETING TUMORS IN RATS--III. ESTABLISHMENT OF IMMUNOGLOBULIN-SECRETING CELL LINES FROM LOU/Wsl STRAIN RATS. (E.) Burtonboy, G. (Fac. Med., Catholic U. Louvain, Belgium), H. Bazin, C. Deckers, A. Beckers, M. E. Lamy and J. F. Heremans. *Eur J Cancer* 9(4):259-262, 1973.

Continuous cell lines were derived from a number of transplantable immunoglobulin-producing tumors of rat origin. Viable tumor cells, obtained by aspirating ascites fluid with a heparinized syringe, were subjected to low-speed centrifugation at room temperature for 10 min. Sedimented cells were washed once with RPMI 1640 Culture medium, then suspended in the same medium supplemented with fetal calf serum. The cultured cells retained the morphological and biosynthetic properties of the original tumor cells. Immuno-electrophoresis showed that concentrated supernatant of one cultured cell line consisted of proteins precipitable by rabbit antiserum to whole rat serum proteins, as well as by rabbit antiserum specific for the heavy chains of the subclass of rat Ig to which the original tumor protein belonged. This cell line secreted 43.9 μ g Ig over a 3-day period. Tumors producing a monoclonal immunoglobulin indistinguishable from that of the original tumor appeared 1-2 wk after i.p. injection of hybrid rats (August/Cen female X LOU/Wsl male) with 25 X 10⁶ cells from culture. The same dose gave rise to tumor nodules when inoculated s.c. into histocompatible animals.

- 3966 ASSOCIATION OF TUMOR AND HISTOCOMPATIBILITY ANTIGENS IN SERA OF LYMPHOMA-BEARING MICE. (E.) Fujimoto, S. (Fac. Med., U. Manitoba, Winnipeg, Canada), C. H. Chen, E. Sabbadini and A. H. Sehon. *J Immunol* 111(4):1093-1100, 1973.

Soluble tumor-associated antigens and histocompatibility antigens were detected in sera of A/J mice bearing a spontaneous lymphoma (L1117) by the inhibition of the cytotoxicity of rabbit anti-L1117 serum and of alloantisera to normal A/J antigens, resp.; the rabbit antiserum was rendered specific for lymphoma antigens by extensive absorption with normal A/J cells and allo-antisera were produced in C57BL/6J and C3H mice. Both types of antigen were isolated together from serum of tumor-bearing animals, with reverse immunosorbents prepared by insolubilization of either rabbit anti-L1117 antibodies or of the anti-A/J alloantibodies, and were also present in the supernatant of the medium used for culturing L1117 cells. It is suggested that the determinants responsible for these two antigenic specificities were associated with the same molecular moiety.

- 3967 THE BIOLOGICAL BEHAVIOUR OF THE TRANSMISSIBLE VENEREAL TUMOUR IN IMMUNOSUPPRESSED DOGS. (E.) Cohen, D. (Dept. Animal Pathol., U. Cambridge, England). *Eur J Cancer* 9(4):253-258, 1973.

The biological behavior of the transmissible venereal tumor of the dog (TVT) was studied in whole body X-irradiated dogs. The tumor showed a regular malig-

nant biological behavior in dogs which had been given 200 rads whole-body X-irradiation before tumor transplantation (4.5 x 10⁷ trypsinized TVC cells, s.c.). In seven of the eight X-irradiated dogs, the TVT became ulcerated and in six dogs it metastasized. In control dogs the tumor showed a more benign and irregular biological behavior which ranged from no tumor growth or growth followed by regression in some dogs, to metastases and ulceration in others. In all dogs in which metastases could be detected they appeared in the inguinal lymph nodes and replaced most of the lymphoid tissue. In three different cases the iliac lymph node, the axillary lymph node and the spleen were also affected. Sera, collected at intervals from experimental and control dogs, were tested for the presence of anti-TVT antibodies using the Indirect Membrane Immunofluorescence Test. Only minor differences in anti-TVT antibody activity could be detected between the two groups.

- 3968 IMMUNOLOGICAL STUDIES OF MEMBRANE GLYCOPROTEINS ISOLATED FROM HUMAN BREAST CARCINOMAS. (E.) Kuo, T.-T. (Washington U. Sch. Med., St. Louis, Mo.), J. Rosai and T. W. Tillack. *Int J Cancer* 12(2):532-542, 1973.

Glycoproteins were isolated from membrane preparations of human breast cancer using lithium di-iodosalicylate followed by phenol partition as described for the isolation of carcinoembryonic antigen (CEA) from colonic carcinoma. The glycoprotein isolated from breast cancer (BCGP) differed from colonic CEA in extraction yield (approximately one-third that of CEA), antigenicity, molecular wt on sodium dodecyl sulfate-acrylamide gel electrophoresis and on immuno-electrophoresis. Rabbit anti-serum raised against BCGP and absorbed with red blood cells and normal breast tissue showed a reaction of identity on Ouchterlony immunodiffusion between BCGP and CEA. However, antiserum to CEA absorbed with red blood cells and normal colonic mucosa showed cross-reaction of BCGP with CEA. The anti-BCGP activity in anti-CEA could be removed by absorption with BCGP, leaving activity directed only against CEA. Therefore, BCGP appears to be a CEA-like antigen that shares antigenic determinants with CEA and may be responsible for the elevated radioimmunoassays for CEA in breast cancer patients. Whether there is a breast-cancer-specific antigen associated with the cell membrane remains to be demonstrated.

- 3969 ALTERNATE CHANGES OF SURFACE ANTIGEN(S) IN ADENOVIRUS TYPE 12-TRANSFORMED AND TUMOR CELLS. (E.) Nakajima, S. (Inst. Virus Res., Kyoto U., Japan), C. Hamada and H. Uetake. *Jap J Microbiol* 17(4):303-311, 1973.

Alternate changes of specific surface antigen(s) (S antigen) were examined in transformed and tumor cells induced by human adenovirus type 12. All of the hamster and mouse cells transformed *in vitro* showed ring form membrane fluorescence staining by anti-S antigen rabbit sera, whereas tumor cells, either induced by the virus *in vivo* or produced by inoculation with

the S(+)-transformed cells, did not show any fluorescence. When the S(-) tumor cells were serially subcultured *in vitro*, all of them converted to S(+) cells, although more than ten subcultures were necessary. For the S(+) cells to form tumors in hamsters about ten times as many cells were necessary as the S(-) cells. This difference became greater when tumor formation was tested in preimmunized hamsters, while little, if any, when tested in X-irradiated hamsters. In addition, immunogenicity of the S(+) cells was suggested to be higher than that of the S(-) cells. These findings indicate that the S(+) cells are more immunosensitive and immunogenic than the S(-) cells, and that *in vivo* conversion from S(+) to S(-) may be due to selection of S(-)-mutant cells. *In vitro* conversion from S(-) to S(+) was also suggested to be due to the appearance of S(+)-mutant cells.

970 EPSTEIN-BARR-VIRUS ANTIBODIES IN AMERICAN AND AFRICAN BURKITT'S LYMPHOMA. (E.) Birshaut, Y. (Sloan-Kettering Inst., New York, N.Y.), H. Cohen and D. A. Stevens. *Lancet* (7821):114-116, 1973.

A comparative study of the distribution of serum antibody against Epstein-Barr (E.B.) virus among 89 African and American patients with Burkitt's lymphoma confirmed the nearly universal presence of antibody to this virus in African cases. However, only one-third of the American patients had detectable E.B.-virus antibody levels. In Africa 77% of age and sex matched control sera were found to have detectable levels of antibody to E.B. virus, while in American controls the frequency of antibody was 47%. Also, African Burkitt's tumor cases generally had markedly raised titers (1/640 or greater), while titers in the American cases did not differ significantly from those found in an age, sex, and race matched control group. There was no correlation between antibody titer and the course of disease in either African or American patients. The findings suggest either that African and American Burkitt's lymphoma are different diseases, or that E.B. virus is unlikely to be the etiological agent of Burkitt's lymphoma in both Africa and America.

71 THE SPECIFICITY OF THE CYTOPLASMIC ANTIGEN IN HUMAN MALIGNANT MELANOMA. (E.) Elliott, G. (Westminster Med. Sch., London, England), B. Jarlow, P. R. G. Needham and M. G. Lewis. *Eur J Cancer* 9(8):607-610, 1973.

Sera from 152 patients with a wide range of pigmented and nonpigmented skin lesions were incubated with melanoma smears, obtained from fresh biopsy specimens snap-frozen in liquid nitrogen-isopentane, in the presence of fluorescein-labeled goat antihuman γ globulin. Antibody to cytoplasmic antigen in melanoma cells could be demonstrated only in sera of two patients with early or localized malignant melanoma. The specificity of cytoplasmic antigen in melanoma cells to raise antibodies in these patients alone indicates the value of serum examination as a screen-test in cases of suspected malignant melanoma.

3972 CARCINOEMBRYONIC ANTIGEN (CEA): A COMPARISON OF THE FARR AND Z-GEL METHODS FOR CEA DETECTION IN BENIGN AND MALIGNANT DISEASES. (E.) Band, P. (U. Alberta, Edmonton, Canada), H. Hyde, M. Feldstein, T. McPherson and V. Patwardhan. *Eur J Cancer* 9(8):597-602, 1973.

Carcinoembryonic Antigen (CEA) was assayed in the serum of 177 patients with the Farr radioimmunoassay and in the plasma of 166 patients with the Z-gel radioimmunoassay. Patients were classified into four groups according to diagnosis: (I: gastrointestinal (GIT) cancer; II: non-GIT cancer; III: benign GIT disease; IV: benign non-GIT disease). Abnormal CEA levels (> 2.5 ng/ml) were obtained in 66%, 57%, 39%, and 15% with the Farr technique, and in 70%, 44%, 47%, and 12% with the Z-gel method in the diagnosis groups I, II, III, and IV resp. The same aliquots of blood were also assayed in 72 patients with both techniques; results agreed in 87% of the cases. Statistical evaluation of the data indicate no difference between the two methods. The range of probability of a false negative CEA value is high no matter which technique is used. This limitation is partially circumvented by raising the considered normal CEA value (2.5 ng/ml) to higher levels (greater than 8 ng/ml).

3973 THE STIMULATING EFFECT OF SOME SERA ON LYMPHOCYTES OF CHRONIC LYMPHATIC LEUKAEMIA *IN VITRO*. (E.) Pössnerova, V. (Hematol. Blood Transfusion Inst., Charles U., Prague, Czechoslovakia), F. Herman-sky, T. Poch and J. Fortynova. *Neoplasma* 20(3):251-254, 1973.

A comparative study of prolonged *in vitro* cultivation without stimulation of normal leukocytes and leukocytes of 28 patients with chronic lymphadenosis (CLL) was performed. The lymphocytes of normal subjects did not change morphologically. The CLL lymphocytes incubated for 14 days with homologous serum showed an increase in size of a number of lymphocytes and/or a partial transformation to blastoid cells with low incidence of mitoses in 81% of experiments. Enlarged cells stained with buffered toluidine blue showed conspicuous nucleoli with a relatively uniform distribution of structures containing ribonucleoproteins. An increase in ^3H -uridine uptake occurred in these samples.

3974 THE LACK OF EFFECT OF MOUSE EPIDERMAL GROWTH FACTOR (EGF) AND ITS ANTI-SERUM ON HUMAN TISSUES. (E.) Waghe, M. (Dept. Child Hlth., U. Manchester, England), S. Kumar and J. K. Steward. *Eur J Cancer* 9(8):603-605, 1973.

Administration of Epidermal Growth Factor (EGF), isolated from adult male mouse submaxillary glands, into newborn mice (2 $\mu\text{g}/\text{k}$ for 7 days) resulted in stunted growth, loss of hair, wrinkling of skin, precocious eruption of incisors and early opening of eyelids, and thickening and keratinization of epidermis. A single precipitation line was obtained in both gel-diffusion and immunoelectrophoresis using rabbit antisera to EGF. Fluorescent-labeled anti-EGF

serum specifically stained tubular nuclei in the basal part of the cells of the mouse submandibular gland. The *in vitro* growth of three normal human kidneys and four normal breasts and of 16 human tumors (9 breast carcinomas, 4 Wilms' tumors, 2 neuroblastomas, and 1 hypernephroma) was not influenced by mouse EGF or its antiserum. Similarly, fluorescein-isothiocyanate-labeled antisera failed to stain any cellular component of the human tissues.

- 3975 ON THE MODE OF ACTION OF BCG. (E.) Mitchell, M. S. (Yale U. Sch. Med., New Haven, Conn.), D. Kirkpatrick, M. B. Mokyr and I. Gery. *Proc Am Assoc Cancer Res* 14(March):4, 1973.
- 3976 ANALYSIS OF ^{125}I -LABELED ANTIGLIOMA ANTIBODIES ADSORBED BY INTACT MONOLAYERS OF CULTURED GLIOMA CELLS. (E.) Day, E. D. (Duke U. Med. Ctr., Durham, N.C.) and D. D. Bigner. *Proc Am Assoc Cancer Res* 14(March):7, 1973.
- 3977 *IN-VIVO* MACROPHAGE INTERACTIONS WITH LYMPHOCYTES IN HODGKIN'S DISEASE. (E.) Massek, M. A. (Dept. Med., Stanford U., Calif.), D. J. Rhoades and J. H. Frenster. *Proc Am Assoc Cancer Res* 14(March):8, 1973.
- 3978 DIFFERENCES BETWEEN MOTHERS OF CHILDREN WITH LEUKEMIA AND MOTHERS OF NORMAL CHILDREN. (E.) Hann, H.-W. L. (Inst. Cancer Res., Philadelphia, Pa.), W. T. London, A. I. Sutnick, H. M. Carim, A. E. Evans and B. S. Blumberg. *Proc Am Assoc Cancer Res* 14(March):9, 1973.
- 3979 IMMUNIZATION AGAINST LEUKEMIA BY SKIN SCARIFICATION IN GUINEA PIGS. (E.) Gross, L. (VA Hosp., New York, N.Y.). *Proc Am Assoc Cancer Res* 14(March):10, 1973.
- 3980 CARCINOEMBRYONIC ANTIGEN IN OSTEOGENIC SARCOMA. (E.) Cortes, E. P. (Roswell Park Mem. Inst., Buffalo, N.Y.), J. J. Wang, E. D. Holyoke and G. P. Murphy. *Proc Am Assoc Cancer Res* 14(March):11, 1973.
- 3981 FAMILIAL STOMACH CANCER AND IMMUNOLOGIC ABNORMALITIES. (E.) Creagan, E. T. (Natl. Inst. Hlth., Bethesda, Md.) and J. F. Fraumeni, Jr. *Proc Am Assoc Cancer Res* 14(March):13, 1973.
- 3982 PLASMA CARCINOEMBRYONIC ANTIGEN (CEA) IN PATIENTS WITH ADVANCED CANCER. (E.) Wiernik, P. H. (Natl. Cancer Inst., Bethesda, Md.) and A. A. Serpick. *Proc Am Assoc Cancer Res* 14(March):14, 1973.
- 3983 ECTOPIC PLACENTAL HORMONES IN NONTROPHOBLASTIC TUMORS: SERIAL MEASUREMENTS FOLLOWING CHEMOTHERAPY. (E.) Muggia, F. M. (Albert Einstein Coll. Med., New York, N.Y.), S. W. Rosen, B. D. Weintraub and H. H. Hansen. *Proc Am Assoc Cancer Res* 14(March):20, 1973.
- 3984 HETEROTRANSPLANTATION STUDY OF HUMAN MELANOMA. (E.) Mukherji, B. (Tufts-New Engl. and Med. Ctr., Boston, Mass.), A. Flowers and L. Nathanson. *Proc Am Assoc Cancer Res* 14(March):40, 1973.
- 3985 IMMUNOLOGICAL CHARACTERISTICS OF HAMSTER CELLS TRANSFORMED BY HERPES SIMPLEX VIRUS TYPE 2. (E.) Doller, E. (M. S. Hershey Med. Ctr., Pennsylvania St. U.), R. Duff and F. Rapp. *Proc Am Assoc Cancer Res* 14(March):40, 1973.
- 3986 COMPLEMENT-DEPENDENT CYTOTOXICITY OF SERUM OF PATIENTS WITH MALIGNANT MELANOMA. (E.) Porta, G. D. (Natl. Tumor Inst., Milan, Italy), G. Fossati and S. Canevari. *Proc Am Assoc Cancer Res* 14(March):33, 1973.
- 3987 EFFECT OF COMPLETE FREUND'S ADJUVANT ON TRANSPLANTED AND SPONTANEOUS LYMPHOMA IN AKR MICE. (E.) Check, J. H. (Hahnemann Med. Coll., Philadelphia, Pa.), L. W. Brady and E. A. O'Neill. *Proc Am Assoc Cancer Res* 14(March):33, 1973.
- 3988 SPECIFIC AND NON-SPECIFIC IMMUNOLOGIC REACTIVITY OF REGIONAL LYMPH NODE LYMPHOCYTES IN HUMAN MALIGNANCY. (E.) Ambus, U. (M. D. Anderson Hosp., Tumor Inst., Houston, Tex.), G. Mavligit, C. McBride and E. Hersch. *Proc Am Assoc Cancer Res* 14(March):34, 1973.
- 3989 RELATIONSHIP OF PHA RESPONSE TO CLINICAL STAGE OF LEUKEMIA IN CHILDREN. (E.) Ciudad, H. (Roswell Park Mem. Inst., Buffalo, N.Y.) and L. F. Sinks. *Proc Am Assoc Cancer Res* 14(March):34, 1973.
- 3990 INDUCTION BY HALOGENATED PYRIMIDINES OF THREE DIFFERENT ANTIGENS ASSOCIATED WITH EPSTEIN-BARR VIRUS (EBV) IN ALL CLONES FROM NON-PRODUCING HUMAN LYMPHOBLASTOID CELL LINES. (E.) Sugawara K. (Hokkaido U. Sch. Med., Sapporo, Japan), F. Mizuno and T. Osato. *Proc Am Assoc Cancer Res* 14(March):36, 1973.
- 3991 MIXED LEUKOCYTE CULTURE (MLC) RESPONSES OF HL-A IDENTICAL SIBLINGS TO ACUTE LEUKEMIC BLASTS. (E.) Anderson, P. N. (Johns Hopkins U. Sch. Med., Baltimore, Md.), W. B. Bias and G. W. Santos. *Proc Am Assoc Cancer Res* 14(March):40, 1973.

- 3992 ELEVATED LEVELS OF SERUM α -FETOPROTEIN IN RATS BEARING HEPATOMAS INDUCED BY AFLATOXIN B₁. (E.) Sontag, J. M. (Natl. Cancer Inst., Bethesda, Md.) and R. Kroes. *Proc Am Assoc Cancer Res* 14(March):43, 1973.
- 3993 EFFECTIVENESS OF THE COMBINED SPECIFIC AND NON-SPECIFIC IMMUNOSTIMULANTS IN DBA/2 MICE. (E.) Holland, J. F. (Roswell Park Mem. Inst., Buffalo, N.Y.), G. St. Arneault and J. G. Bekesi. *Proc Am Assoc Cancer Res* 14(March):44, 1973.
- 3994 IMMUNOGLOBULIN COMPLEXES IN PATIENTS WITH MALIGNANCY. (E.) Samayoa, E. A. (Mayo Fdn., Rochester, Minn.), F. C. McDuffie and A. M. Nelson. *Proc Am Assoc Cancer Res* 14(March):48, 1973.
- 3995 IMMUNOLOGIC INHIBITION OF CELLULAR PROLIFERATION IN TUMOR ALLOGRAFTS. (E.) Kreider, J. W. (Coll. Med., Pennsylvania St. U., Hershey, Pa.) and J. W. Combs. *Proc Am Assoc Cancer Res* 14(March):50, 1973.
- 3996 ANTIBODY RESPONSE TO TUMOR SPECIFIC CELL SURFACE ANTIGEN AND CROSS REACTIVE ANTIGENS IN TUMOR CELLS. (E.) Ting, C. C. (Natl. Inst. Hlth., Bethesda, Md.) and R. B. Herberman. *Proc Am Assoc Cancer Res* 14(March):51, 1973.
- 3997 DETECTION OF HUMAN ANTISARCOMA ANTIBODIES BY IMMUNODIFFUSION. (E.) Winters, W. D. (U. California, Los Angeles, Sch. Med.) and D. L. Morton. *Proc Am Assoc Cancer Res* 14(March):55, 1973.
- 3998 NON-SPECIFIC RESISTANCE TO TUMOR GROWTH IN LISTERIA IMMUNE MICE. (E.) Youdim, S. (U. Minnesota, Minneapolis), M. Moser and O. Stutman. *Proc Am Assoc Cancer Res* 14(March):58, 1973.
- 3999 *IN VITRO* INDUCTION OF TUMOR-SPECIFIC TRANSPLANTATION ANTIGEN IN MOUSE CELLS ABORTIVELY INFECTED WITH PAPOVAVIRUS SV40. (E.) Tevethia, S. S. (Baylor Coll. Med., Houston, Tex.) and V. L. McMillan. *Proc Am Assoc Cancer Res* 14(March):57, 1973.
- 4000 ANTIGENS SPECIFIC FOR HUMAN LYMPHOCYTIC OR MYELOGENOUS LEUKEMIA. (E.) Metzgar, R. S. (Duke U. Med. Ctr., Durham, N.C.), T. Mohanakumar and D. S. Miller. *Proc Am Assoc Cancer Res* 14(March):49, 1973.
- 4001 IMMUNOLOGICAL STUDIES IN BALB/C MICE WITH AN EXPERIMENTALLY INDUCED TUMOR. (E.) Bhatnagar, R. M. (Mount Sinai Med. Sch., New York, N.Y.), A. R. Rausen and J. B. Zabriskie. *Proc Am Assoc Cancer Res* 14(March):48, 1973.
- 4002 MALIGNANCY IN INDIVIDUALS WITH PRIMARY IMMUNODEFICIENCY DISEASES: THE IMMUNODEFICIENCY-CANCER REGISTRY. (E.) Kersey, J. H. (U. Minnesota Med. Sch., Minneapolis), B. D. Spector and R. A. Good. *Proc Am Assoc Cancer Res* 14(March):57, 1973.
- 4003 THE EFFECT OF HYPERIMMUNE ENHANCING ANTISERUM ON CELL MEDIATED IMMUNITY. (E.) Cohen, J. M. (Natl. Cancer Inst., Bethesda, Md.). *Proc Am Assoc Cancer Res* 14(March):59, 1973.
- 4004 THE EFFECT OF MYCOPLASMA CONTAMINATION OF CELL CULTURES ON THE RESULTS OF A COMPLEMENT-MEDIATED CYTOTOXICITY TEST. (E.) Bloom, E. T. (U. California, Los Angeles). *Proc Am Assoc Cancer Res* 14(March):61, 1973.
- 4005 *IN VIVO* ROLE OF LYMPHOCYTE DEPENDENT ANTIBODY IN MEDIATING TUMOR ALLOGRAFT REJECTION. (E.) Zighelboim, J. (Sch. Med., U. California, Los Angeles), B. Bonavida and J. L. Fahey. *Proc Am Assoc Cancer Res* 14(March):61, 1973.
- 4006 PREFERENTIAL GROWTH OF C3Hf MOUSE LUNG TUMORS IN (C3Hf x A)_{F1} HYBRID MICE: IMMUNOLOGIC CROSS REACTIONS BETWEEN TUMORS AND NORMAL STRAIN A LUNG TISSUE. (E.) Cotton, W. G. (Natl. Cancer Inst., Bethesda, Md.), J. M. Rice and E. Esber. *Proc Am Assoc Cancer Res* 14(March):63, 1973.
- 4007 THE FATE OF NEURAMINIDASE-TREATED LEUKEMIA L1210 CELLS AS AN IMMUNOGEN IN NON-IMMUNIZED MICE. (E.) Bekesi, J. G. (Roswell Park Mem. Inst., Buffalo, N.Y.), L. Walter and J. F. Holland. *Proc Am Assoc Cancer Res* 14(March):64, 1973.
- 4008 ANTIBODIES IN HUMAN SERA TO THE MAMMALIAN ONCORNAVIRUS INTERSPECIES ANTIGEN. (E.) Olsen, R. G. (Ohio St. U., Columbus) and D. S. Yohn. *Proc Am Assoc Cancer Res* 14(March):67, 1973.
- 4009 CELL MEDIATED IMMUNITY IN DORMANT FRIEND LEUKEMIA VIRAL INFECTIONS. (E.) Wheelock, E. F. (Jefferson Med. Coll., Philadelphia, Pa.), R. H. Kerman, J. Clark and S. T. Toy. *Proc Am Assoc Cancer Res* 14(March):68, 1973.
- 4010 EVALUATION OF THREE CEA ASSAYS. (E.) Fleisher, M. (Mem. Hosp., Sloan-Kettering Inst., New York, N.Y.), E. Besenfelder, M. K. Schwartz and H. F. Oettgen. *Proc Am Assoc Cancer Res* 14(March):68, 1973.
- 4011 INCREASING THE IMMUNOGENICITY OF "PRIVATE" MAMMARY TUMOR ANTIGENS WITH NEURAMINIDASE. (E.) Simmons, R. L. (U. Minnesota, Minneapolis) and A. Rios. *Proc Am Assoc Cancer Res* 14(March):70, 1973.

- 4012 IMMUNIZATION AGAINST MAMMARY TUMORIGENESIS IN MICE WITH FORMALIN-INACTIVATED MTV. (E.) Charney, J. (Inst. Med. Res., Camden, N.J.), D. H. Moore, J. A. Holben and C. M. Cody. *Proc Am Assoc Cancer Res* 14(March):71, 1973.
- 4013 REDUCED TUMORIGENICITY OF SYNGENEIC MOUSE SARCOMA CELLS RESISTANT TO ACTINOMYCIN D AND ETHIDIUM BROMIDE. (E.) Biedler, J. L. (Sloan-Kettering Inst., New York, N.Y.) and R. H. F. Peterson. *Proc Am Assoc Cancer Res* 14(March):72, 1973.
- 4014 CORRELATION OF IMMUNE RESPONSE TO PROGRESSIVE TUMOR AND PROTECTIVE VACCINATION. (E.) Prager, M. D. (U. Texas Southwestern Med. Sch., Dallas) and J. Ribble. *Proc Am Assoc Cancer Res* 14(March):74, 1973.
- 4015 TRANSFER OF TUMOR IMMUNITY IN GUINEA PIGS WITH RNA EXTRACTS. (E.) Paque, R. (U. Illinois, Med. Ctr., Chicago), B. Zbar, M. Meltzer, S. Dray and H. Rapp. *Proc Am Assoc Cancer Res* 14(March):78, 1973.
- 4016 CELL-MEDIATED IMMUNE RESPONSES OF CANCER PATIENTS TO CANDIDA ANTIGEN. (E.) Maigetter, R. (Mount Sinai Hosp. Med. Ctr., Chicago, Ill.), E. Ezdinli, R. A. Smith and N. J. Bigley. *Proc Am Assoc Cancer Res* 14(March):78, 1973.
- 4017 USE OF NORMAL ANTIGEN BLOCKING SERA TO DIRECT THE SPECIFICITY OF ANTISERA TOWARDS MOUSE LEUKEMIA CELLS. (E.) Reif, A. E. (Tufts U. Sch. Med. Surg. Service, Boston, Mass.), P. J. Smith and C. M. Robinson. *Proc Am Assoc Cancer Res* 14(March):80, 1973.
- 4018 CARCINOEMBRYONIC ANTIGEN (CEA) ASSOCIATED WITH RAT MAMMARY CANCER AND ITS RELATION TO IMMUNOGENICITY AND METASTASIZING CAPACITY. (E.) Kim, U. (Roswell Park Mem. Inst., Buffalo, N.Y.) and M. Tunis. *Proc Am Assoc Cancer Res* 14(March):80, 1973.
- 4019 IDENTIFICATION OF T- AND B-LYMPHOCYTES IN HUMAN LYMPHATIC LEUKEMIA. (E.) Minowada, J. (Roswell Park Mem. Inst., Buffalo, N.Y.), T. Han and T. Ohnuma. *Proc Am Assoc Cancer Res* 14(March):81, 1973.
- 4020 MEDIATION OF IMMUNE RESPONSES TO MURINE TUMORS BY "IMMUNE" RNA. (E.) Linker-Israeli, M. (U. California, Los Angeles) and Y. H. Pilch. *Proc Am Assoc Cancer Res* 14(March):82, 1973.
- 4021 IMMUNOFLUORESCENT STUDIES OF HUMAN MALIGNANT MELANOMA. (E.) Wood, G. W. (U. Kansas Med. Ctr., Kansas City) and R. F. Barth. *Proc Am Assoc Cancer Res* 14(March):22, 1973.
- 4022 STUDIES OF STABILITY OF CARCINO-EMBRYONIC ANTIGEN IN PATIENT'S PLASMA. (E.) Meeker, W. R., Jr. (U. Kentucky, Coll. Med., Lexington), R. Kashmiri, L. Hunter and W. O. Griffen, Jr. *Proc Am Assoc Cancer Res* 14(March):83, 1973.
- 4023 DEMONSTRATION OF FETAL ANTIGENS IMMUNOLOGICALLY SIMILAR TO TUMOR ANTIGENS IN GUINEA PIG AND MURINE METHYLCHOLANTHRENE INDUCED TUMORS. (E.) Wells, S. (Natl. Cancer Inst., Bethesda, Md.), J. Grant and B. LeMevel. *Proc Am Assoc Cancer Res* 14(March):83, 1973.
- 4024 NATURAL CYTOTOXIC REACTIVITY OF RAT LYMPHOCYTES AGAINST SYNGENEIC GROSS LEUKEMIA. (E.) Nunn, M. (Natl. Cancer Inst., Bethesda, Md.), J. Djeu, D. Lavrin and R. Herberman. *Proc Am Assoc Cancer Res* 14(March):87, 1973.
- 4025 CHIMPANZEE RESPONSE TO IMMUNIZATION WITH HUMAN MELANOMA CELLS. (E.) Hornung, M. O. (Tulane U. Sch. Med., New Orleans, La.), S. P. Leong, C. A. Sutherland and E. T. Krementz. *Proc Am Assoc Cancer Res* 14(March):92, 1973.
- 4026 INDUCTION OF IMMUNITY WITH INACTIVATED GROSS LEUKEMIA VIRUS (GLV) AND THE ROUTES OF TRANSMISSION TO PROGENY. (E.) Keller, S. E. (Lenox Hill Hosp., New York, N.Y.), M. L. Gimovsky and H. L. Joachim. *Proc Am Assoc Cancer Res* 14(March):98, 1973.
- 4027 ANTIBODIES TO EPSTEIN-BARR VIRUS (EBV)-ASSOCIATED ANTIGENS IN FAMILIAL CANCER. (E.) Levine, P. H. (Natl. Inst. Hlth., Bethesda, Md.), J. Fraumeni, J. I. Reisher and D. Waggoner. *Proc Am Assoc Cancer Res* 14(March):98, 1973.
- 4028 SIALIC ACID AND THE "MASKING" OF FETAL ANTIGENS. (E.) Hannon, W. H. (U. Tennessee, Knoxville), N. G. Anderson and J. H. Coggin, Jr. *Proc Am Assoc Cancer Res* 14(March):98, 1973.
- 4029 ANATOMICAL REGIONAL DIFFERENCES IN THE IMMUNE CONTROL OF INDUCED METASTASES. (E.) Vaage, J. (Massachusetts Gen. Hosp., Boston). *Proc Am Assoc Cancer Res* 14(March):70, 1973.
- 4030 THE ENHANCEMENT OF ANTITUMOR ANTIBODY BINDING TO CROSS REACTIVE NORMAL TISSUE ANTIGENS - A COMPLEMENT MEDIATED EFFECT. (E.) Drake, W. P. (Natl. Inst. Hlth., Baltimore, Md.) and M. R. Mardiney, Jr. *Proc Am Assoc Cancer Res* 14(March):84, 1973.
- 4031 EFFECT OF ANTI-TUMOR IMMUNITY ON PREGNANCY IN THE MOUSE. (E.) Parmiani, G. (Natl. Tumor Inst., Milan, Italy). *Proc Am Assoc Cancer Res* 14(March):32, 1973.

- 4032 QUANTITATION OF SERUM ALPHA-FETOPROTEIN IN MONKEYS WITH PRIMARY LIVER TUMORS. (E.) McIntire, K. R. (Nat'l. Cancer Inst., Bethesda, Md.), G. L. Princler and R. H. Adamson. *Proc Am Assoc Cancer Soc* 14(March):104, 1973.
- 4033 BLASTOGENIC RESPONSE OF NORMAL HUMAN LYMPHOCYTES TO ALLOGENEIC LEUKEMIC CELLS. (E.) Han, T. (Roswell Park Mem. Inst., Buffalo, N. Y.). *Proc Am Assoc Cancer Res* 14(March):104, 1973.
- 4034 CHARACTERIZATION OF ANTIBODY TO CARCINO-EMBRYONIC ANTIGEN (CEA) IN HAMSTERS XENOGRAFTED WITH A HUMAN COLONIC TUMOR. (E.) Primus, F. J. (Hoffman-La Roche, Inc., Nutley, N.J.), R. H. Wang, H. J. Hansen and D. M. Goldenberg. *Proc Am Assoc Cancer Res* 14(March):105, 1973.
- 4035 AN ANTIGENIC MARKER FOR HUMAN MELANOCYTES. (E.) Smith, R. W. (Nat'l. Inst. Hlth., Bethesda, Md.). *Proc Am Assoc Cancer Res* 14(March):105, 1973.
- 4036 CARCINOEMBRYONIC ANTIGEN IN PATIENTS WITH CARCINOMA OF THE LUNG. (E.) Vincent, R. G. (Roswell Park Mem. Inst., Buffalo, N.Y.) and T. M. Chu. *Proc Am Assoc Cancer Res* 14(March):106, 1973.
- 4037 ^{51}Cr RELEASE CELLULAR LYMPHOCYTE CYTOTOXICITY AS A POSSIBLE MEASURE OF IMMUNOLOGICAL COMPETENCE OF CANCER PATIENTS. (E.) McCoy, J. (Bionetics Res. Lab., Bethesda, Md.), R. Herberman, E. Perlin, P. Levine and C. Alford. *Proc Am Assoc Cancer Soc* 14(March):107, 1973.
- 4038 THYMO-LYMPHATIC ORGAN RESPONSE TO THE LDH-VIRUS. (E.) Santisteban, G. (Pacific Northwest Res. Fdn., Seattle, Wash.) and V. Riley. *Proc Am Assoc Cancer Res* 14(March):112, 1973.
- 4039 CYTOTOXIC ANTIBODY TO AUTOLOGOUS LYMPHOCYTES IN CHILDHOOD LEUKEMIA. (E.) Dreesman, G. R. (Baylor Coll. Med., Houston, Tex.), D. L. Brill, D. J. Fernbach and M. Benyesh-Melnick. *Proc Am Assoc Cancer Res* 14(March):85, 1973.
- 4040 IMMUNOGENIC PROPERTIES OF A SOLUBILIZED TUMOR ANTIGEN FROM AN RNA VIRUS-TRANSFORMED NEOPLASM. (E.) Appella, E. (Nat'l. Cancer Inst., Bethesda, Md.), T. Fischetti and L. W. Law. *Proc Am Assoc Cancer Res* 14(March):113, 1973.
- 4041 IMMUNOLOGICAL CROSSREACTIVITY OF TUMOR ASSOCIATED FETAL ANTIGENS. (E.) Salinas, F. A. (Carcinogenesis Program, Oak Ridge Nat'l. Lab., Tenn.) and M. G. Hanna, Jr. *Proc Am Assoc Cancer Res* 14(March):116, 1973.
- 4042 AUTOGENOUS IMMUNITY TO ENDOGENOUS RNA TUMOR VIRUS: CHRONIC HUMORAL IMMUNE RESPONSE TO VIRAL ENVELOPE ANTIGENS IN B6C3F1 MICE. (E.) Batzing, B. L. (Carcinogenesis Program, Oak Ridge Nat'l. Lab., Tenn.), J. N. Ihle, M. Yurconic, R. W. Tennant and M. G. Hanna, Jr. *Proc Am Assoc Cancer Res* 14(March):116, 1973.
- 4043 ALPHA-FETOPROTEIN IN SERA OF PATIENTS WITH GASTROINTESTINAL AND TESTICULAR TUMORS. (E.) Kithier, K. (Dept. Oncology, Wayne St. U., Detroit), V. K. Vaitkevicius, S. J. Figiel, J. Cejka and M. Al-Sarraf. *Proc Am Assoc Cancer Res* 14(March):117, 1973.
- 4044 MIXED LYMPHOCYTE REACTIVITY (MLR) OF PATIENTS WITH SOLID TUMORS. (E.) Harris, J. (Ottawa Gen. Hosp., Canada), D. Copeland, D. Hyslop and T. Stewart. *Proc Am Assoc Cancer Res* 14(March):117, 1973.
- 4045 OCCURRENCE OF FLUORESCENT AND PRECIPITIN ANTIBODIES TO A BOVINE C-TYPE VIRUS (BLV) AMONG THE CATTLE POPULATION. (E.) Ferrer, J. F. (New Bolton Ctr., U. Pennsylvania, Kennett Square) and D. Bhatt. *Proc Am Assoc Cancer Res* 14(March):118, 1973.
- 4046 EVIDENCE ASSOCIATING ESP-1 CELL LINE WITH HUMAN TUMOR POPULATIONS. (E.) Priori, E. S. (U. Texas, M. D. Anderson Hosp., Tumor Inst., Houston), L. Dmochowski and J. R. Wilbur. *Proc Am Assoc Cancer Res* 14(March):118, 1973.
- 4047 IMMUNOPROPHYLAXIS (IP) OF MALIGNANT MELANOMA (MM) WITH BCG. (E.) Gutterman, J. U. (M. D. Anderson Hosp., Tumor Inst., Houston, Tex.). *Proc Am Assoc Cancer Res* 14(March):119, 1973.
- 4048 SURFACE ANTIGENS OF CULTURED HUMAN SARCOMAS DETECTED BY IMMUNE ADHERENCE TEST. (E.) Irie, K. (U. California, Los Angeles, Sch. Med.) and R. F. Irie. *Proc Am Assoc Cancer Res* 14(March):119, 1973.

See also:

- * (Rev): 3601, 3621, 3622, 3624, 3635, 3637, 3639
- * (Chem): 3656, 3679, 3681, 3685, 3699
- * (Viral): 3809, 3812, 3814, 3816, 3822, 3823, 3836, 3851, 3856, 3876, 3884, 3904, 3909

- 4049 **DYSERYTHROPOIESIS, REFRACTORY ANEMIA, AND "PRELEUKEMIA": METABOLIC FEATURES OF THE ERYTHROCYTES.** (E.) Valentine, W. N. (Departments Med., Pediatrics, Path., U. California, Los Angeles), P. N. Konrad and D. E. Paglia. *Blood* 41(6):857-875, 1973.

Twenty-one enzymatic activities and red cell glutathione content were compared in cord blood, dyserythropoietic disorders, normal subjects, and subjects with hemolytic syndromes and reticulocytosis approximating that of the newborn. The 28 dyserythropoietic states observed were heterogeneous and included several hereditary disorders, "preleukemia" and overt leukemia, refractory anemia with and without marrow ringed sideroblasts, and folate deficiency. Many activities exceeded those of cord erythrocytes and "high reticulocyte" controls by one or even several standard deviations. The low phosphoglycerate kinase and glutathione peroxidase and relatively low ribosophosphate pyrophosphokinase and adenine-phosphoribosyl transferase of cord erythrocytes were mimicked uncommonly. Pyruvate kinase was often relatively or absolutely low and sometimes dramatically so. Enzyme ratios were grossly aberrant. Capricious very high individual activities occurred in some instances. The heterogeneous nature of case material, including both hereditary and acquired syndromes, renders it difficult to interpret similarities often seen from case to case and sometimes to cord blood patterns on the basis of the reversion to fetal erythropoiesis as a common denominator. Rather, the cyto- and karyokinetic abnormalities characterizing dyserythropoiesis of diverse etiologies may result in exaggerations and distortions of a normal asynchronism in loss of functional genetic material governing enzyme synthesis as the nucleus degenerates and cytoplasmic organelles are lost.

- 4050 **CYCLIC LEUKOCYTOSIS IN CHRONIC MYELOGENOUS LEUKEMIA: NEW PERSPECTIVES ON PATHOGENESIS AND THERAPY.** (E.) Gatti, R. A. (Departments Pediatrics, Path., Lab. Med., U. Minnesota, Minneapolis), W. A. Robinson, A. S. Deinard, M. Nesbit, J. J. McCullough, M. Ballow and R. A. Good. *Blood* 41(6):771-782, 1973.

Studies were performed on a teen-age female identical twin with cyclic leukocytosis and Ph¹-positive chronic myelogenous leukemia (CML), in an attempt to analyze the relationship of these cycles to her disease and to utilize the cycles as reference points for kinetic studies and as landmarks in the progression of her disease. She remained untreated for 2½ yr. Colony-stimulating factor levels showed an inverse relationship to the peripheral leukocyte count. Two distinct phases in cyclic patterns of peripheral leukocyte counts prior to treatment could be appreciated in retrospect: phase 1, a stable 15-month period during which leukocyte counts peaked every 10 wk and then returned to baseline levels, and phase 2, an 18-month period of insidious deterioration during which the low points of successive cycles became progressively higher and leukocytes were accumulating in the peripheral blood at a rate of 400 X 10⁶/day. It is proposed that the

increased marrow production of myeloid cells in this disease does not represent a life-threatening situation until cell accumulation begins.

- 4051 **COELIAC DISEASE AND MALIGNANCY.** (E.) Barry, R. E. (Dept. Med., U. Bristol, England) and A. E. Read. *Q J Med* 42(168):665-675, 1973.

Studies of small-bowel epithelial-cell turnover were performed on 18 cases diagnosed as having adult celiac disease. All patients were fed a gluten-free diet. The 10 who responded to this diet showed a significant change toward normality in tests of absorptive function and in the histologic appearance of their jejunal biopsy. In the eight patients who did not respond to exclusion of gluten from their diet, the DNA loss rate of the duodenal mucosa and the total mucosal thickness were significantly decreased compared with the gluten-responsive patients. Mitotic indexes were also decreased in the nonresponsive group, but the difference was not statistically significant. These results suggest that patients who fail to respond to treatment with a gluten-free diet have a hypoplastic mucosa. Of the eight nonresponders, five have since died from lymphosarcoma (2), reticulosarcoma (2), and focal malignancy in mesenteric nodes (1). The failure of eight patients to respond to dietary gluten exclusion does not exclude a diagnosis of celiac disease but does suggest that the disease process producing mucosal architectural changes is different from that of uncomplicated celiac disease. More sophisticated methods are needed to identify those patients likely to develop malignancy.

- 4052 **LEVELS OF CYCLIC AMP AND CYCLIC AMP PHOSPHODIESTERASE DURING DIFFERENTIATION OF MOUSE NEUROBLASTOMA CELLS IN CULTURE.** (E.) Prasad, K. N. (U. Colorado Med. Ctr., Denver) and S. Kumar. *Proc Am Assoc Cancer Res* 14(March):6, 1973.

- 4053 **PLATELET SURVIVAL AND ADENOSINE TRIPHOSPHATASE (ATP-ASE) ACTIVITY IN AKR MICE.** I. (E.) Brodsky, I. (Hahnemann Med. Coll., Philadelphia, Pa.) and N. V. Dimitrov. *Proc Am Assoc Cancer Res* 14(March):8, 1973.

- 4054 **THE PATHOGENESIS OF ALVEOLAR CELL CARCINOMA INDUCED BY DIBUTYLNITROSAMINE IN BUFFALO RATS.** (E.) Levin, S. (U. Chicago, Ill.). *Proc Am Assoc Cancer Res* 14(March):24, 1973.

- 4055 **HETEROTRANSPLANTATION OF CANINE MAMMARY TISSUES TO HAMSTER CHEEK POUCH.** (E.) Richmond, R. E. (U. California, Davis) and L. J. Faulkin. *Proc Am Assoc Cancer Res* 14(March):62, 1973.

- 4056 **POTENTIAL PRECANCEROUS LESIONS IN THE HUMAN BREAST.** (E.) Wellings, S. R. (Sch. Med., U. California, Davis) and H. M. Jensen. *Proc Am Assoc Cancer Res* 14(March):76, 1973.

4057 STUDIES ON THE MECHANISM OF SKIN TUMOR PROMOTION. THE EARLY EPIDERMAL REACTION TO TUMOR PROMOTERS, CARCINOGENS AND HYPERPLASTIC AGENTS. (E.) Raick, A. N. (Dept. Path., U. Toronto, Canada). *Proc Am Assoc Cancer Res* 14(March): 82, 1973.

4058 CYTOGENETIC DIFFERENCES BETWEEN NORMAL AND LEUKEMIC HUMAN BLOOD LYMPHOID CELL LINES DURING THE COURSE OF THEIR ESTABLISHMENT. (E.) Venaut, A.-M. (Paul-Brousse Hosp., Villejuif, France) and C. Rosenfeld. *Proc Am Assoc Cancer Res* 14(March):101, 1973.

4059 CONTROL OF NEUTROPHILIC GRANULOCYTOPOIESIS. (E.) Bierman, H. R. (Inst. Cancer, Blood Res., Beverly Hills, Calif.) and J. E. Hood. *Proc Am Assoc Cancer Res* 14(March):127, 1973.

See also:

- * (Rev): 3601, 3618, 3625
- * (Chem): 3663
- * (Viral): 3825, 3847, 3885

- 4060 CANCER IN ITS GEOGRAPHICAL PERSPECTIVE. (E.) Doll, R. (Radcliffe Infirmary, Oxford, England), M. S. R. Hutt, D. J. Jussawalla, D. P. Burkitt and J. Higginson. *Proc Royal Soc Med* 66(4):307-318, 1973.

Geographical environment, socio-economic or cultural influences on the basic patterns and incidence of cancer are reviewed. In India the upper digestive tract (oral cavity, pharynx and esophagus) and the uterine cervix are most frequently affected. Chewing of pan quid (betel leaves) is an important factor in initiating oral cancer, whereas smoking tends to lead to pharyngeal cancers. Multiple pregnancies starting from an early age represent an important risk factor in those Indian women who ultimately develop cervical cancer. Tobacco plays an important role in the etiology of esophageal cancer in India. A link between alcohol and esophageal cancer is apparent only in association with other factors such as smoking and chewing of tobacco. The site distribution of cancer in India is almost at complete variation with the distribution seen in the West. In Africa cancers of the lung, colon, rectum, body of the uterus and breast have a low frequency while cancers of the liver, cervix and choriocarcinoma have a higher frequency than in Europe. Burkitt's lymphoma and epithelioma, which arises in the often healed scars of tropical leg ulcers, are prevalent exclusively in equatorial countries. Stomach cancer, Kaposi's sarcoma and esophageal cancer exhibit foci of occurrence within East Africa, the latter two being much less prevalent in West Africa. Within East Africa distribution of Burkitt's lymphoma related to temperature, rain, and the distribution of hyperendemic malaria. The distribution of penile cancer is strongly influenced by circumcision habits, although variations are shown between tribes who do not practice circumcision. In conclusion, the general benefits, potential developments, and limitations of geographical pathology are reviewed. Epidemiological studies could clarify areas of chemical carcinogenesis and be of value in separating and identifying risks. The author previously cited evidence that 80% of tumors in North America and Europe are conditioned by the present environment and thus are theoretically preventable.

- 4061 LUNG CANCER IN WOMEN: PRESENT AND FUTURE TRENDS. (E.) Wynder, E. L. (American Hlth. Fdn., New York, N.Y.), L. S. Covey and K. Mabuchi. *J Natl Cancer Inst* 51(2):391-401, 1973.

One argument against the causative relationship between cigarette smoking and lung cancer has been that mortality for U.S. females remains low, despite a large number of female smokers. However, the male:female ratio, which showed virtual unity at the early part of this century, has steadily increased in the U.S. The ratio rose from 1.7 in 1930 to a highest level of 6.6 in 1960. Since the dramatic rise of lung-cancer mortality in females in 1960, the sex ratio decreased to 6.0 in 1965 and 5.1 in 1969. The present retrospective study of 108 female lung-cancer patients seen during 1970-72 showed that cigarette smoking was closely as-

sociated with epidermoid and oat-cell types of lung cancer, and less strongly with glandular types of lung cancer. Among cigarette smokers of >1 pack/day, a longer history of smoking, earlier starting age, inhalation, and nonfilter cigarette smoking were significantly associated with greater lung-cancer morbidity ratios. Another study of 1839 male and 2213 female control patients showed great differences in various smoking-intensity factors between men and women, but the differences diminished at younger ages. As women adopt cigarette-smoking habits similar to those of men, the death rate from lung cancer in women will continue to increase. However, the level is not expected to attain that presently observed in men, since young women began smoking cigarettes with lower tar-yield (i.e., filters) than did men presently of lung-cancer age.

- 4062 EPIDEMIOLOGY OF MALIGNANT NEOPLASMS IN NICKEL REFINERIES. (Rus.) Saknyn', A. V. (Inst. Industrial Hyg. Occupational Health, Sverdlovsk, USSR) and N. K. Shabynina. *Gig Tr Prof Zabol* (9):25-29, 1973.

Statistical analysis of death rates for lung cancer, stomach cancer, and sarcomas showed that these were significantly higher among workers in four nickel refineries in the Urals than in the populations of the towns in which these refineries were located. Death rates for lung cancer were particularly high among workers in roasting and reduction plants who were exposed to aerosols of nickel sulfide, oxide, and metallic nickel; in workers in electrolysis plants where work was done with high concentrations of nickel sulfate and nickel chloride in the air; in workers in smelting plants who were exposed to dust from nickel ores; in workers in cobalt refineries where the most toxic substance was cobalt dust; and in workers who came into contact with arsenic. Death rates for stomach cancer were increased in workers engaged in all types of occupations. Workers who died of lung cancer had worked an average of 7-13 yr in the refinery, and those who died of stomach cancer, 10-14 yr. The high incidence of stomach cancer might be due to direct action of nickel on the gastric mucosa since nickel dust is readily soluble in acid media. Death rates for sarcoma among nickel workers were 1.8-6.2 times higher than those of the populations of the towns in which the refineries were located. Most men dying of sarcoma were over 40 yr old. The most frequent sites of sarcomas were in the hip, lungs, and intestine. Since the highest death rates for sarcomas were found in nickel refineries which had treated nickel cobalt concentrates containing arsenic, sarcomas are attributed to arsenic.

- 4063 AGGREGATION OF HODGKIN'S DISEASE. (E.) Lee, Y.-T. N. (Ellis Fischel State Cancer Hosp., Columbia, Mo.). *J Am Med Wom Assoc* 28(10):529-535, 1973.

During a retrospective and all-inclusive study of 190 patients with the diagnosis of Hodgkin's disease at the Ellis Fischel State Cancer Hospital from 1940

to 1971, family history of Hodgkin's disease was recorded in two pairs of siblings. All four cases (one pair of brothers and one pair of sisters) came from the same small Missouri town. No interlinking factors are evident between the two families. The literature was reviewed for reported Hodgkin's disease cases in immediate and distant family members, in non-familial close contacts and cases clustered in time and/or space by statistical studies. At present these epidemiological studies provide no consistent conclusion and offer no definite answer regarding the etiology of Hodgkin's disease.

4064 EPIDEMIOLOGICAL FACTORS IN LUNG CANCER AMONG WOMEN IN EL PASO COUNTY, TEXAS 1944-1969.

(E.) MacDonald, E. J. (U. Texas System Cancer Ctr., Houston), H. Lichtenstein, D. Nooner, D. Flory, S. Wikstrom and J. Oro. *J Amer Womens Assoc* 28(9):459-467, 1973.

El Paso County, Texas, has had an extensive cancer follow-up program which has been prospective since 1950, and thus offers the opportunity for study of environmental influences on cancer of specific sites in Latins and Anglos sharing the same physical environment. The Latin women were found to be a high incidence group. A number of etiological factors were found, the most important of which was residence from time of birth to adult life in an adobe house. These houses are nearly airtight, poorly ventilated and heated usually by burning of oil, wood, coal or other fuel burned in the center of the room. Scrapings from the walls and ceilings were analyzed and polynuclear aromatic hydrocarbons and DDT were found. The other variables besides details of dwelling place included lifetime exposures to dust storms, copper smelter smoke, heating materials, occupational exposure, disease history, economic status, and smoking history by type, amount and duration.

4065 PROLIFERATIVE PROPERTIES OF MALIGNANT CELL POPULATIONS CULTURED IN INTRAPERITONEAL DIFFUSION CHAMBERS. (E.) Laerum, O. D. (Max Planck Inst. Virus Res. Tubingen, W. Germany), A. Gruneisen and M. F. Rajewsky. *Eur J Cancer* 9(8):533-541, 1973.

The proliferative characteristics of four different types of malignant cells cultured in an i.p. diffusion chamber (ID chamber) were investigated, and compared with the corresponding data obtained under conditions of cell culture *in vitro* and growth *in vivo*. The cell types were BICR/MIR_k cells, a permanent malignant cell culture line originating from a transplantable mammary tumor of the Marshall rat; Fortner A Mel 1 cells, a transplantable malignant melanoma of the golden hamster in the ascitic form; a transplantable ethylnitrosourea-induced leukemia (L5222) of the BD IX rat; and finally leukemic cells from two human patients with untreated acute myeloblastic leukemia. After an initial decline in the cell number, all cell types investigated proliferated in ID-chambers implanted into BD IX rats, for periods of 8-13 days. The tumor cell populations grew exponentially for 2-5 days, with an actual doubling time corresponding to that obtained during growth *in vivo*. The human

leukemic cells behaved as a "steady-state" system, where the rate of proliferation was apparently compensated by a corresponding rate of cell death. The rate of proliferation in ID-chambers was slower than under cell culture conditions *in vitro*. Ten min after i.v. injection of hydroxyurea (HU) into the host animals, the HU concentration in the ID-chambers had equilibrated with the blood concentration of HU, when measured on the third day after implantation of the chambers. On the 7th day, the rate of diffusion of the compound between the blood and the chambers was slower, probably due to an inflammatory reaction on the external side of the chambers. The mode of proliferation of the investigated cell populations during diffusion chamber culture may be more representative of the normal growth conditions *in vivo* than of those in cell culture *in vitro*.

4066 PRIMARY LIVER CANCER IN CALIFORNIA. (E.) Krain, L. S. (U. California Med. Ctr., Los Angeles). *Oncology* 28(2):117-125, 1973.

Appropriate data from 1,277 primary liver cancer cases first diagnosed in 58 participating hospitals and reported in the California Tumor Registry and related sources were examined in order to clarify various epidemiologic and survival facts about the occurrence of this cancer in relation to a survey of the world's literature. The age-adjusted mortality rates for primary liver cancer in nonwhites have increased in California due to a high immigrant population of Chinese and Japanese, while the white rates have remained relatively constant. Liver cancer occurs in excess in lower socioeconomic group populations, California Chinese and Japanese and at a median age of 61.8 years. A male sex preponderance (male/female ratio of 1.8:1) suggests certain occupationally related variables may be basic to the etiology of liver cancer. There has been a slight absolute, but a large percentage increase in the relative one and five year survival rates for liver cancer (primary) due to a greater percentage of patients being diagnosed when localized and a greater percentage undergoing palliative or curative surgery. The 5 yr relative survival rates for all stages are 3% and for localized cases 10%.

4067 THE RELATIONSHIP BETWEEN GROWTH RATE, LABELLING INDEX AND HISTOLOGICAL TYPE OF HUMAN SOLID TUMOURS. (E.) Malaise, E. P. (Inst. Gustave-Roussy, Villejuif, France), N. Chavaudra and M. Tubiana. *Eur J Cancer* 9(4):305-312, 1973.

Published data on the labeling index of 156 human solid tumors, incubated *in vitro* with tritiated thymidine, were pooled with laboratory results for 86 tumors. Mean values for five histologic groups were calculated. For embryonal tumors and hemato-sarcomas, the mean value of the labeling index (L.I.) is about 30% and appears to correspond to a growth fraction (G.F.) close to 100%. The L.I. values for the other three groups are significantly lower, being 8.3% for squamous cell sarcomas, 3.8% for mesenchymal sarcomas, and 2.1% for adenocarcinomas.

The L.I. and doubling time of these five groups are negatively correlated. Of all the parameters of the kinetics of tumor cell proliferation, the G.F. varies most among different histologic types and is the most highly correlated with the previously estimated growth rate. The cell loss factor varies less from one histologic group to another and is highest in groups with the highest G.F. In spite of very high rates of cell loss in embryonal tumors and hematosarcomas, this parameter does not by itself explain the differences in growth rates of the various groups.

- 4068 NEPHROBLASTOMA: INDEX CANCER OF CHILDHOOD.
(E.) Innes, M. D. (Princess Alexandra Hosp., Brisbane, Australia). *Med J Aust* 2(7):322-323, 1973.

The incidence of malignant kidney tumors in children to the age of 14 yr was compared in all those registries recording their results in both Volumes 1 and 2 of "Cancer Incidence in Five Continents". It was found that the incidence of these tumors did not vary significantly between registries ($\chi^2_{(14)} = 18.8720$; $P > 0.1$). This finding was expected on the assumption that most childhood cancers are hereditary genetic disorders in Hardy-Weinberg equilibrium; as such, both the relative frequency and incidence of any particular malignant disease can be expected to vary between ethnic groups, since the ethnic genome controls both incidence and relative frequency of all traits in Hardy-Weinberg equilibrium. Nephroblastomas, being embryonic tumors and hence arising before the action of specifically racial genes, were expected to be uninfluenced by the ethnic genome.

- 4069 CANCER OF THE PROSTATE IN OSLO 1958-1967.
(E.) Fryjordet, A. (Ullevål Hosp., Oslo, Norway), A. Ekeland, A. Moe, S. Ous and G. Waaler. *J Oslo City Hosp* 23(8):129-137, 1973.

Of the 496 patients treated for cancer of the prostate during the last 10 yr in the Oslo City Hospital, 91% received hormonal treatment. Thirty-one percent survived after 5 yr. More than half of the deaths were caused by the cancer and nearly one third by cardiovascular diseases. The series shows a high frequency of cardiovascular complications and an excess mortality from cardiovascular diseases probably associated with estrogen therapy. The indications for estrogen treatment should be limited and the dosage reduced. Radiation therapy may be of value in some cases. The main problem is to diagnose the cancer in an early stage in which radical prostatectomy is possible.

- 4070 CARCINOMA OF THE VULVA IN CALIFORNIA 1942-1969. THE CALIFORNIA TUMOR REGISTRY EXPERIENCE. (E.) Krain, L. S. (U. California Med. Ctr., Los Angeles). *Oncology* 28(2):110-116, 1973.

An analysis of vulvar carcinoma (ICD No. 176.0) from the California Tumor Registry data, 1942-1969, was performed. While the incidence rate for vulvar cancer

has increased slightly from 1.8 (1960) to 2.2 (1969) per 100,000 population (age-adjusted), the absolute and relative survival rates for vulvar carcinoma in California for localized lesions have declined drastically (85% 5 yr relative survival in 1942-1954 versus 75% 5 yr relative survival in 1955-1969), and localized lesions have increased from 7 to 23% for these time periods, resp. The reason for this decline appears to be a decline in the use of radical vulvectomy, radiation therapy and adjuvant radiation therapy. For all stages of vulvar cancer, the 5-yr relative survival rates have increased from 66% (1942-1954) to 70% (1955-1969). A further study of these data by the 5th International Congress of Obstetrics and Gynecologists (Sidney, Australia, 1967) classification scheme is in progress. The rarity of vulvar cancer in the nonwhite races is emphasized.

- 4071 INCORPORATION OF MACROMOLECULAR PRECURSORS INTO HEPATOMAS. (E.) Lea, M. A. (New Jersey Med. Sch., Newark), F. L. Khalil and H. P. Morris. *Proc Am Assoc Cancer Res* 14(March):11, 1973.

- 4072 STUDY OF FAST (F) AND SLOW (S) GROWING TRANSPLANTABLE TUMORS DERIVED FROM SPONTANEOUS MAMMARY TUMORS (SMT) OF THE DBA/2Ha-DD MOUSE. (E.) Hosokawa, M. (Roswell Park Mem. Inst., Buffalo, N.Y.), F. Orsini and E. Mihich. *Proc Am Assoc Cancer Res* 14(March):39, 1973.

- 4073 INFLUENCE OF AGE ON THE GROWTH OF A TRANSPLANTED TUMOR AS SHOWN IN SINGLE AND PARABOTICALLY UNITED YOUNG AND OLD RATS. (E.) Han, P. Y. (Dept. Anatomy, McGill U., Montreal, Canada), N. J. Nadler and C. P. Leblond. *Proc Am Assoc Cancer Res* 14(March):56, 1973.

- 4074 CELL KINETICS AND TUMOR GROWTH RATE CHARACTERISTICS OF DIFFERENT MORRIS HEPATOMAS. (E.) Looney, W. B. (U. Virginia Sch. Med., Charlottesville), A. A. Mayo, M. Mitchell and H. P. Morris. *Proc Am Assoc Cancer Res* 14(March):72, 1973.

See also:

- * (Rev): 3606, 3628
* (Immun): 3978, 4002

- 4075 PERIODIC ACID SCHIFF POSITIVE MYELOBLASTS IN CHRONIC MYELOGENOUS LEUKAEMIA: RELATION TO KARYOTYPE EVOLUTION. (E.) Pedersen, B. (Danish Cancer Soc., Aarhus). *Scand J Haematol* 11(2): 112-121, 1973.

The relationship between periodic acid-Schiff (PAS)-positive chronic myelogenous leukemia (CML) myeloblasts and the cytogenetic characters of granulocyte precursors was examined. In 20 venous blood samples from 11 patients with Ph¹-positive CML the frequencies of PAS-positive myeloblasts were determined. Karyotype analysis was performed after *in vitro* culture of the samples. The frequencies of circulating PAS-positive myeloblasts were positively correlated (1) to the relative size of the myeloblastic compartment, (2) to the frequencies of metaphases containing additional chromosomes, and (3) in particular to those showing excess C chromosomes. These observations suggest that premature development of PAS positivity reflects a defective granulocyte precursor differentiation which is due to evolution of certain types of aneuploid clones.

- 4076 MULTIPLE MYELOMA IN CHILDHOOD: REPORT OF A CASE WITH BREAST TUMORS AS A PRESENTING MANIFESTATION. (E.) Maeda, K. (Muhlenberg Hosp., Plainfield, N.J.), C. M. Abesamis, L. M. Kuhn and B. H. Hyun. *Am J Clin Pathol* 60(4):552-558, 1973.

A case of multiple myeloma of the IgA monoclonal gammopathy type is reported in a 13-year-old Caucasian female. Unusual presenting manifestations included s.c. nodules of the left neck and right upper abdomen and recurrent nodules of both breasts. The lesions had caused no discomfort but grew progressively. Urinalysis showed plus 2 albuminuria, but tests for Bence Jones protein, cryoglobulin and pyroglobulin were negative as was also the Sia test. She showed an initial response to cyclophosphamide and allopurinol therapy. Tumor nodules recurred, however, and radiotherapy and prednisone were added to the regimen. The patient developed a falsely-positive heterophile antibody. A course of vincristine was given, but the patient complained of increasing abdominal and chest pain and had spiking fever. The patient finally died of bronchopneumonia and other pulmonary complications secondary to therapy. Autopsy revealed extensive, widespread disease involving breasts, ovaries, pancreas, spleen, liver, pleura, peritoneum, skin, lymph nodes, GI tract, pituitary and bone marrow. The entire course of the disease lasted only three months.

- 4077 UNUSUAL ASPECTS OF CERVICAL CANCER. (E.) Ford, J. H., Jr. (U. Miami Sch. Med., Fla.), R. C. Dudan and H. E. Averette. *Gynecol Oncol* 1(2): 123-129, 1973.

Four case histories are presented which represent some unusual clinical aspects of cervical cancer. The first case is that of a 52-yr-old Negro female who presented with irregular vaginal bleeding of

five months duration. Biopsy of a sessile polypoid ectocervical mass showed invasive squamous cell carcinoma. Cone biopsy revealed no residual cervical cancer. At operation for total abdominal hysterectomy, metastatic disease was discovered which failed to respond to radiation therapy. Autopsy revealed widespread pelvic disease and multiple liver and lung nodules. The second case, a 51-yr-old asymptomatic Negro female, showed a class III routine Pap smear. Seven yr previously a cone biopsy for another class III smear had revealed *in situ* carcinoma which was not treated by hysterectomy. At the time of referral, punch biopsy revealed invasive cervical squamous cell carcinoma. At operation for radical hysterectomy, metastatic disease was found in the periaortic, common iliac, external iliac and hypogastric chains on the left side. In spite of no further therapy, the patient is clinically free of disease after eight yr. The third case is that of a 35-yr-old White female with a class I Pap smear and an asymptomatic endocervical polyp. Biopsy of the polyp, which was expelled spontaneously, revealed invasive endocervical adenocarcinoma. Radical hysterectomy was performed and the patient is clinically free of disease after two yr. The last case is that of a 35-yr-old White female with a two yr history of invasive squamous cell carcinoma who had refused treatment. Repeated Pap smears had, however, been class I until one showed class IV cells. Punch biopsy revealed invasive squamous cell carcinoma. Radical hysterectomy was performed and the patient is clinically free of tumor two yr later.

- 4078 STIMULATION OF OLIGODEOXYRIBONUCLEOTIDE SYNTHESIS BY CYTOPLASMIC FACTORS IN THE ISOLATED NUCLEI OF LEUKEMIC CELLS. (E.) Schandl, E. K. (Nova U., Life Sci. Ctr., Fort Lauderdale, Fla.). *Cancer Res* 33(10):2398-2401, 1973.

Nuclei were isolated by hypotonic shock and homogenization from freshly drawn, nonstimulated rat leukemic leukocytes and were pulse labeled with deoxythymidine triphosphate-³H. Labeled oligodeoxyribonucleotides were separated from the bulk of DNA and proteins by membrane cone filtration and were further purified by alkaline hydrolysis and diethylaminoethyl cellulose chromatography. In parallel experiments, cytoplasmic factors (CF), obtained after removal of nonsoluble material from the cytoplasm by high-speed centrifugation, were included. More than a twofold increase of label incorporation into oligodeoxyribonucleotides was observed. The effect was greatest when fresh CF were used; either storage at -68 C or freezing and thawing reduced the stimulatory effect.

- 4079 ADENOCARCINOMA OF THE UTERINE CERVIX. (E.) Kagan, A. R. (S. California Permanente Kaiser Fdn. Hosp., Los Angeles), H. Nussbaum, P. Y. M. Chan and H. K. Ziel. *Am J Obstet Gynecol* 117(4):464-468, 1973.

The survival rates of 30 female patients presenting with stage I and II cervical adenocarcinoma and

treated primarily by irradiation were studied. Radiation therapy consisted of 3,000 rad megavoltage applied in the midplane to the pelvis followed by two radium applications of 3,000 mg hr each. The overall five-yr survival rate for these patients was 50% (58% for stage I and 40% for stage II). Overall 10 yr survival was 33%. After five yr, most patients who died did so secondary to distant metastases rather than to local pelvic disease as was common before five yr. The five yr survival rate reported in the literature for patients undergoing radiation plus hysterectomy was 80%. Thus, in spite of a higher incidence of serious complications in patients receiving combined therapy, it was felt that such a regimen should be considered as being more effective than radiation alone in controlling cervical adenocarcinoma.

- 4080 COMPARATIVE STUDIES ON THE *IN VITRO* UPTAKE OF ^3H -CYTIDINE AND ^3H -URIDINE BY NORMAL AND LEUKAEMIC LYMPHOCYTES. (E.) Bremer, K. (Ctr. Basic Clin. Res., U. Ulm, Germany), W. Schreml and E. B. Harriss. *Scand J Haematol* 11(2):122-130, 1973.

The influence of lymphocyte concentration and of temperature on the intensity of lymphocyte labelling was studied in normal and leukemic cell populations. A desired level of cell concentration was found to be 2×10^4 to 2×10^5 and the desired temperature 37 C. Normal leukocytes showed a fairly constant behavior with respect to total uptake of radioactivity, incorporation into intracellular fractions and the ratio of ^3H -cytidine to ^3H -uridine incorporation (average ratio 10 ± 1). In contrast, the blood lymphocytes of patients with chronic lymphocytic leukemia (CLL) exhibited a great variation in the parameters studied, ranging from below normal to several times normal values. This broad spectrum of *in vitro* characteristics has to be taken into consideration when populations of CLL lymphocytes are compared to normal lymphocytes and to those of other lymphatic disorders.

- 4081 PROSTAGLANDIN PRODUCTION BY NEUROBLASTOMA, GLIOMA AND FIBROBLAST CELL LINES; STIMULATION BY N^6 , O^2 -DIBUTYRYL ADENOSINE 3':5'-CYCLIC MONOPHOSPHATE. (E.) Hamprecht, B. (Max Planck Inst. Biochem., Munich, W. Germany), B. M. Jaffee and G. W. Philpott. *FEBS Letters* 36(2):193-198, 1973.

The capability of a neuroblastoma, a glioma, and a fibroblast cell line to synthesize prostaglandin E (PGE) was investigated. The cell lines used were N4TG3, a 6-thioguanine resistant mutant of mouse neuroblastoma clone N4, C6-BU-1, a bromodeoxyuridine resistant mutant of rat glioma clone C 6, and B82, a bromodeoxyuridine resistant mutant L cell line. For prostaglandin determination, cell homogenates were extracted with a mixture of 2 ml H_2O , 5 ml ethylacetate and 3 ml of a 3:3:1 mixture of ethylacetate:isopropanol:0.1 M HCl. The prostaglandins were separated by chromatography on silicic acid and assayed by a radioimmunochemical method. At

day 3 PGE production/million cells/day was 58 PG for N4TG3, 148 PG for C6-BU-1, and 162 PG for B82. In the presence of dibutyryl cyclic AMP, the values increased to 324 PG for N4TG3, 167 PG for C6-BU-1, and 1252 PG for B82. In all cases PGE production increased with an increase in cell density only in the presence of dibutyryl cyclic AMP. The drug markedly reduced the growth rates of the cell lines and caused morphological changes in glioma cells. Only minor amounts of PGF and PGA were detected in the three cell lines. These results indicate that cyclic AMP plays a positive regulatory function in PG synthesis in the cells investigated.

- 4082 BOUND FORMS OF NUCLEAR DNA POLYMERASE IN REGENERATING AND NEOPLASTIC RAT LIVERS.

(E.) Chiu, J.-F. (M. D. Anderson Hosp. Tumor Inst., Houston, Texas), C. Craddock and L. S. Hnilica. *FEBS Lett* 36(2):235-238, 1973.

The activity of solubilized, bound DNA polymerase (3-4S) increased considerably after partial hepatectomy and in the livers of male Sprague-Dawley rats fed a diet containing 0.06% *N*, *N*-di-methyl-*p*-(*m*-tolylazo)-aniline ($3'$ -MDAB). Enzyme activity, assayed by the method of Chiu and Sung, began to increase after 18 days on $3'$ -MDAB and peaked (25.92 pmoles/mg protein/min) at day 40. After partial hepatectomy, DNA polymerase activity began to increase at about 18 hr and peaked (35.88 pmoles/mg protein/min) at 48 hr. A high molecular wt DNA polymerase (6-8S) also appeared in rats exposed to $3'$ -MDAB for 24 days; this enzyme peaked around 32 days and disappeared after 104 days exposure to the carcinogen. Partial purification of the DNA polymerases was accomplished by ammonium sulfate fractionation and DEAE-cellulose column chromatography. The 3-4S and 6-8S enzymes differed in template specificity, Mg^{2+} requirements, and responses to monovalent ion concentrations. The 6-8S enzyme, which is similar to reverse transcriptase, was highly active with poly AU and poly A \cdot poly templates while 3-4S DNA polymerase had almost no activity with these templates. The former enzyme had maximum activity in 10 mM MgCl_2 compared with the 15-25 mM MgCl_2 required for the maximum activity of the 3-4S enzyme. The activity of the lower molecular wt enzyme increased about 200% in the presence of 30 mM KCl as against only 50% for the high molecular wt enzyme.

- 4083 DISTRIBUTION OF LACTATE DEHYDROGENASE AND ITS SUBUNITS IN RAT TISSUES AND TUMORS.

(E.) Schweitzer, E. S. (Harvard Med. Sch., Boston, Mass.), F. Farron and W. E. Knox. *Enzyme* 14(3):173-184, 1973.

Total lactate dehydrogenase activity and the fractions of its M and H subunits were measured in rat tissues and tumors. Except in the case of mammary gland measurements, the rats used were all adult males, 90-100 days old and weighing about 300 g. Fetuses were taken at 18.5-19.5 days of gestation. Highest total activities were found in skeletal and heart muscles and in liver. Second only to these were the high values found in tumors. Immature and

undifferentiated tissues contained only small fractions of the H subunit, which increased with age to high fractions in the differentiated tissues of heart, brain, thymus and kidney. Tumors were among the tissues with the lowest fractions of H subunit. The fraction of H subunit in all tumors was substantially lower than that in the parent normal tissue (or it was low in both, as in skeletal muscle and rhabdomyosarcoma). A commonality among all four different kinds of tumors examined (Morris renal cell carcinoma, Morris hepatoma, rhabdomyosarcoma, and Walker mammary carcinoma) was this increased expression of the M gene, normally most active in immature or undifferentiated tissues.

- 4084 SOME EPR SIGNALS IN TUMOUR TISSUE. (E.) Dodd, N. J. F. (Christie Hosp., Manchester, England). *Br J Cancer* 28(3):257-262, 1973.

Normal and tumor tissues from rats, blood from normal and tumor bearing rats, and normal human blood were examined using the electron paramagnetic resonance (epr) technique. At low temperature a triplet epr signal, which is known to be produced by a NO-hemo-protein complex, was detected in some tumor samples and in decaying normal liver. At room temperature all of the tumor samples examined gave a doublet signal. This signal was also detected in blood but not in other normal tissues. The signal has a g value of 2.0054 ± 0.0002 and a hyperfine splitting of 1.80 ± 0.05 G and is assigned to the ascorbyl free radical. Model experiments suggest that the appearance of detectable concentrations of this radical result from a disturbance of the normal state of the ascorbic acid, dehydroascorbic acid redox system. It was verified that cell division is not responsible for the ascorbyl radical although autolysis may be involved. The formation of ascorbyl radicals in tumor tissue may result from reactions of ascorbic acid, some of which give NO, which in turn may be responsible for formation of the paramagnetic NO-complexes observed.

- 4085 BIOSYNTHESIS OF STEROIDS FROM CHOLESTEROL- ^{14}C BY HUMAN ADRENAL CARCINOMA TISSUE. (E.) Nishida, S. (Kyushu Dental Coll., Fukuoka, Japan), R. A. Jungmann and J. S. Schweppe. *Steroids* 22(3):337-350, 1973.

Slices prepared from human adrenal carcinoma tissue obtained from a 51-yr-old female with virilism were incubated with cholesterol- $4\text{-}^{14}\text{C}$ in the presence and absence of ACTH or cyclic AMP. The tissue homogenates were analyzed for steroids by the reverse isotope dilution method. Purification and identification of the radioactive metabolites were achieved by column, paper and thin-layer chromatography, derivative formation and crystallization to constant specific activity. Cholesterol- ^{14}C was converted to pregnenolone- ^{14}C (0.75%), progesterone- ^{14}C (0.17%) and dehydroepiandrosterone- ^{14}C (0.15%). No evidence was found for the formation of labeled 17-hydroxyprogesterone, androstenedione, testosterone, or estrogens. Addition of ACTH to the incubations resulted in a two-fold increase of radioactive pregnenolone- ^{14}C and or dehydroepiandrosterone- ^{14}C . A two-fold in-

crease of progesterone- ^{14}C synthesis was found in cyclic AMP-stimulated incubations.

- 4086 COMPETITIVE DNA-RNA HYBRIDIZATION OF NUCLEAR AND MICROSOMAL RNA IN NORMAL, NEOPLASTIC, AND NEONATAL LIVER TISSUE. (E.) Garrett, C. T. (U. Wisconsin Med. Sch., Madison), R. E. Moore, C. Katz and H. C. Pitot. *Cancer Res* 33(10):2469-2475, 1973.

The nuclear and microsomal RNA of three minimal deviation hepatomas and 10-day-old neonatal rat liver were compared by standard DNA-RNA competitive hybridization techniques. Nuclear RNA from 5123C hepatoma and 10-day neonatal liver lack sequences present in adult liver nuclear RNA. These missing sequences appear to be identical in part. Nuclear RNA from the slowly growing Morris 9618 hepatoma could not be distinguished from adult liver nuclear RNA. The microsomal RNA from this tumor and the 7800 hepatoma both showed sequences to be missing which were present in adult liver microsomal RNA, whereas microsomal RNA from the Morris 5123C hepatoma could not be distinguished from adult liver microsomal RNA in relation to their competitive efficiency for liver nuclear RNA. The results may be interpreted to support two hypotheses: 1) that alterations in nuclear to cytoplasmic transport of RNA are important in neoplastic transformation, or alternatively 2) that changes in the stability of cytoplasmic microsomal RNA templates may contribute to or be the basis for the neoplastic transformation of cells.

- 4087 TRANSFER RNA METHYLASE AND TRANSFER RNA METHYLASE-INHIBITOR ACTIVITY IN NORMAL AND MALIGNANT HUMAN OVARIAN TISSUE. (E.) Sheid, B. (State U. New York, Downstate Med. Ctr., Brooklyn), T. Lu and J. H. Nelson, Jr. *Cancer Res* 33(10):2518-2523, 1973.

Eight different human ovarian carcinomas were shown to have 2 to 25 times higher transfer RNA methylase-specific activity and transfer RNA methylating capacity than do normal ovarian tissues. The more rapidly metastasizing, poorly differentiated carcinomas had higher transfer RNA methylase activity than did slower metastasizing, well-differentiated, and intermediately differentiated carcinomas. The poorly differentiated carcinomas incorporated from 865 to 2845 pmoles $^{14}\text{CH}_3/\text{mg tRNA}$ and from 205 to 685 pmoles $^{14}\text{CH}_3/\text{mg tRNA/mg protein/hr}$. The transfer RNA methylase activities of all the ovarian tissues, normal and malignant, were directly related to the activity of an endogenous transfer RNA methylase inhibitor: the higher the enzyme activity, the lower the inhibitor activity. Experiments showed that the intermediately differentiated carcinomas and the normal ovarian tissue had the following analogous biochemical characteristics. They produced similar methylation patterns with an exogenous transfer RNA substrate, and they were unable to methylate their own endogenous transfer RNA or that of the others. The poorly differentiated carcinomas differed from normal ovarian tissue in that they synthesized greater percentages of methyl-

ated guanine residues compared with the other methylated bases, contained barely detectable amounts of an endogenous transfer RNA methylase inhibitor, and were capable of methylating transfer RNA isolated from normal ovarian tissue but not their own endogenous transfer RNA.

4088 MANGANESE STIMULATES ADHESION AND SPREADING OF MOUSE SARCOMA I ASCITES CELLS. (E.)

Rabinovitch, M. (New York U. Sch. Med., N.Y.) and M. J. DeStefano. *J Cell Biol* 59(1):165-176, 1973.

Adhesion of Sarcoma I cells (SaI) to untreated or to serum-treated glass was examined by layering ^{51}Cr -labeled cells on the substrate for 20 min at 34 C and determining the glass-bound radioactivity after the monolayers were rinsed. Adhesion to untreated glass proceeded in sodium chloride-imidazole-potassium medium (SIK) without added divalent cations, whereas SaI adhered maximally to the serum-coated substrate only in the presence of 50 μM or more Mn. Divalent Mg, Ca, Co, Ni, or Zn were inactive or minimally active. Mn-stimulated adhesion was sharply temperature dependent, reversible upon removal of Mn, and inhibited by Ca as well as by cytochalasin B, vinblastine, or tetracaine. Adhesion of SaI in SIK did not ensue when cells or the coated substrate were pretreated with Mn and washed in SIK before the adhesion assays. Microscope observations showed that Mn induced the formation of cell processes, ruffles, and veils, and that SaI spread on the uncoated or serum-coated substrate when exposed to Mn. Cells withdrew veils and processes and rounded up when postincubated in Mn-free medium. Formation of cell processes and spreading was inhibited by cytochalasin B, vinblastine, or tetracaine. Manganese-induced adhesion seems to require the participation of microtubules and microfilaments and may be mediated by an effect of Mn on Ca fluxes. The results support the role of cell processes and spreading in cell-to-substrate adhesion.

4089 ORGAN-SPECIFIC DIFFERENCES IN THE METHYLATION OF TRANSFER RNA *IN VITRO*. (E.) Leboy, R. S. (Sch. Dental Med., U. Pennsylvania, Philadelphia) and P. Piester. *Cancer Res* 33(10):2241-2246, 1973.

The methylation of tRNA was studied *in vitro* using an EDTA-treated ammonium sulfate fraction from the 105,000 x g supernatant of rat liver and spleen homogenates. Assay for methylation of various nucleosides using S-adenosylmethionine-methyl- ^{14}C and unfractionated methyl-deficient *E. coli* tRNA or *E. coli* formylmethionine tRNA as substrates in the presence of polyamines revealed differing patterns for liver and spleen, with spleen forming relatively greater amounts of 1-methyladenosine and 7-methylguanosine. Differences were also observed when the methylation reaction was carried out in the absence of polyamines. While enzyme preparations from liver which had been dialyzed against EDTA showed no methylase activity, similar preparations from spleen continued to produce a small amount of 1-methyladenosine and 5-methylcytidine. The observed organ-specific differences in methylating ability could be eliminated by purification on

DEAE-Sephadex. This, plus an increased capacity to methylate *E. coli* formylmethionine tRNA after purification, suggested that the organ-specific differences were due to the presence of inhibitory material. Addition of polyamines to the unpurified assay mixture had partially annulled this inhibitory effect.

4090 MOBILITY OF CARBOHYDRATE-CONTAINING STRUCTURES ON THE SURFACE MEMBRANE AND THE NORMAL DIFFERENTIATION OF MYELOID LEUKEMIC CELLS TO MACROPHAGES AND GRANULOCYTES. (E.) Inbar, M. (Dept. Genet, Weizmann Inst. Sci., Rehovot, Israel), H. Ben-Bassat, E. Fibach and L. Sachs. *Proc Natl Acad Sci USA* 70(9):2577-2581, 1973.

Clones (D^+) of a cultured line of myeloid leukemic cells from SL mice can be induced to undergo normal differentiation to mature macrophages and granulocytes. Other clones (D^-) derived from the same cell line could not be induced to differentiate. The carbohydrate-binding protein concanavalin A (Con A) was used as a probe to study the mobility of carbohydrate-containing sites on the surface membrane of these cells. Changes in the distribution of Con A binding sites on the surface membrane can be induced by Con A. With the appropriate site mobility, this induction of a new distribution resulted in a concentration of Con A-membrane site complexes on one pole of the cell to form a cap. D^+ and D^- clones showed 50 and 5% of cells with caps, resp., although both types of cells bound a similar number of Con A molecules. Treatment of cells with trypsin increased cap formation from 5 to 40% in D^- cells, but did not change the percentage of cells with caps in D^+ cells. The results show a difference in the mobility of Con A binding sites in these two types of cells and suggest a difference in the fluid state of these carbohydrate-containing structures on the surface membrane. It is suggested that a gain of the ability of myeloid leukemic cells to undergo normal differentiation is associated with an increase in the fluidity of structures on the surface membrane where the Con A sites are located. Differences in fluidity of specific membrane sites may also explain differences in the response of cells to other differentiation-inducing stimuli.

4091 HIGH CHROMOSOME NUMBERS OF SEMINOMATA AND MALIGNANT TERATOMATA OF THE TESTIS: A REVIEW OF DATA ON 103 TUMOURS. (E.) Atkin, N. B. (Mount Vernon Hosp., Northwood, Middlesex, England). *Br J Cancer* 28(3):275-279, 1973.

Cytogenetic data on 103 seminomas and malignant teratomas of the testis from the literature and (partly in the form of DNA measurements) from the author's laboratory show that modal chromosome numbers are generally 50 or more. The only exceptions were two seminomas in which diploid and pseudodiploid karyotypes respectively were found, but the dividing cells may not have been tumor cells. Malignant tumors of the testis thus differ from those of all other sites (including the ovary) that have been studied sufficiently, where hypodiploid tumors are common. The reason for this difference is unknown. Mechanisms whereby high chromosome numbers, particularly the near-triploid

numbers commonly found in testicular tumors, may be achieved are repeated non-disjunctions, a combination of a complete doubling of the complement by endoreduplication and chromosomal loss, or "triploidization", which would involve duplication of a haploid or near-haploid set in a diploid or near-diploid cell.

- 4092 LIPID-CHEMICAL DIFFERENCES BETWEEN HUMAN CANCEROUS AND ADENOMATOUS POLYPOUS TISSUES IN THE LARGE INTESTINE. (E.) Nakazawa, I. (Tohoku U. Sch. Med., Sendai, Japan), S. Yamagata and H. Watanabe. *Tohoku J Exp Med* 110(1):23-32, 1973.

Thirteen cases of cancer and ten cases of adenomatous polyp in the large intestine were studied to clarify the biochemical differences between malignant neoplastic and benign growth. Cancerous or adenomatous polypous tissues were collected by biopsy or surgical operation. Total lipid was extracted from each tissue. One part of each total lipid fraction was separated into triglyceride and phospholipid fractions by thin-layer chromatography. The fatty acid composition and the fatty acid content of each lipid fraction were measured by gas-liquid chromatography. When the fatty acid composition of the phospholipid is considered in the form of the deviation rate, the deviation rate of C_{14:0} is remarkably increased and that of C_{20:4} remarkably decreased in the cancerous cases, compared with the respective values of the cases of adenomatous polyp. More definite differences were recognized between the cancerous cases and the cases of adenomatous polyp, when the ratio of the deviation rate of C_{14:0} to that of C_{20:4} was calculated in each case. Namely, these ratios distributed between 2.404 and 4.125 in seven cancerous cases, and in eight cases of adenomatous polyp between 0.564 and 1.856. Thus, if this ratio is used it would be possible to distinguish the cancerous tissue from the adenomatous polypous tissue.

- 4093 ULTRASTRUCTURAL COMPARISON BETWEEN PHYTO-MITOTIC TRANSFORMED NORMAL AND CHRONIC LYMPHOCYTIC LEUKEMIA LYMPHOCYTES. (E.) Douglas, S. D. (Mount Sinai Sch. Med., New York, N.Y.), G. Cohnen and G. Brittinger. *J Ultrastruct Res* 44(1-2):11-26, 1973.

The ultrastructural features of lymphocytes stimulated *in vitro* with phytohemagglutinin (PHA), pokeweed mitogen (PWM), and concanavalin A (Con A) from 12 normal individuals and 18 patients with chronic lymphocytic leukemia (CLL) were studied. With PHA and Con A the transformed cells had characteristics of blasts, and with PWM both blast cells and plasma-cytoid cells occurred. The mean total cell area, the nuclear and cytoplasmic size, and the number of lysosomes were diminished in mitogen-transformed CLL cells as compared to transformed normal lymphocytes. Some stimulated CLL lymphocytes were indistinguishable from normal cells. The results suggest that the CLL lymphocytes which are transformed by phyto-mitogens could be derived from defective leukemic cells and/or a residual population of normal T and B cells.

- 4094 ATAXIA-TELANGELECTASIA. CLONAL GROWTH OF TRANSLOCATION LYMPHOCYTES. (E.) Hecht, F. (U. Oregon Med. Sch., Portland), B. K. McCaw and R. D. Koler. *N Engl J Med* 289(6):286-291, 1973.

- 4095 ADENOCARCINOMA OF THE COLON ASSOCIATED WITH URETEROSIGMOIDOSTOMY: REPORT OF A CASE. (E.) Tank, E. S. (U. Michigan Med. Ctr., Ann Arbor.), D. N. Karsch and J. Lapides. *Dis Colon Rectum* 16(4):300-304, 1973.

- 4096 ADENOSQUAMOUS CARCINOMAS OF THE VULVA AND VAGINA. (E.) Rhatigan, R. M. (U. Hosp. Jacksonville, Fla.) and Q. Mojadidi. *Am J Clin Pathol* 60(2):208-217, 1973.

- 4097 CHROMOSOMAL PATTERNS IN MYELOCYTIC LEUKEMIA. (E.) Rowley, J. D. (Franklin McLean Mem. Res. Inst., Chicago, Ill.). *N Engl J Med* 289(4):220-221, 1973.

- 4098 MURAMIDASE ACTIVITY IN LEUKEMIA AND MYELOPROLIFERATIVE DISORDERS. (E.) Skarin, A. T. (Peter Bent Brigham Hosp., Boston, Mass.), Y. Matsuo and W. C. Moloney. *Oncology* 27(5):406-414, 1973.

- 4099 FIBROSARCOMA METASTATIC TO THE BRAIN. AN UNUSUAL CASE. (E.) Dal Canto, M. (Albert Einstein Coll. Med., Bronx, N.Y.) and M. P. Yalsamis. *Arch Pathol* 96(2):108-110, 1973.

- 4100 ISOLATION AND CHARACTERIZATION OF CELL MITOCHONDRIA FROM AN AH 130 STRAIN OF YOSHIDA HEPATOMA ASCITES. (It.) De Montalvo, A. (Queen Helen Inst. Rome, Italy), E. Piccolella, A. Frigola, G. Pepe and D. Guerriore. *Boll Soc Ital Biol Sper* 48(9):233-236, 1972.

- 4101 SEPARATION OF REVERSE TRANSCRIPTASE FROM DNA-DEPENDENT DNA POLYMERASE. ANALYSIS OF A DNA SYNTHETIZED ON AN RNA TRANSFORMING MODEL. (Fr.) Beljanski, M. (Pasteur Inst. Paris, France). *C R Acad Sci [D] (Paris)* 276(10):1625-1628, 1973.

- 4102 ELECTRON MICROSCOPY FINDINGS IN DIFFERENT ENDOCRINE ACTIVITY ADENOMAS OF THE PARATHYROID. (Ger.) Thiele, J. (Hannover Med. Sch. Inst. Path., W. Germany), E. Reale and A. Georgii. *Virchows Arch [Zellpathol]* 12(2):168-188, 1973.

- 4103 ESTROGEN METABOLISM IN PATIENTS AT HIGH RISK FOR ENDOMETRIAL CARCINOMA. II. THE ROLE OF ANDROSTENEDIONE AS AN ESTROGEN PRECURSOR IN POSTMENOPAUSAL WOMEN WITH ENDOMETRIAL CARCINOMA. (E.) Hausknecht, R. U. (Mount Sinai Sch. Med., New York, N. Y.) and S. B. Gusberg. *Am J Obstet Gynecol* 116(7):981-984, 1973.

- 4104 NUCLEAR DNA CONTENT OF LOBULAR CARCINOMA *IN SITU* OF THE BREAST. (E.) Ludwig, A. S. (Columbia U. Coll. Physicians, Surg., New York, N.Y.), T. Okagaki, R. M. Richart and R. Lattes. *Cancer* 31(6):1553-1560, 1973.
- 4105 INTRAMURAL ADENOFIBROMA OF THE FALLOPIAN TUBE. LIGHT AND ELECTRON MICROSCOPY. (E.) Kanbour, A. I. (U. Pittsburgh Sch. Med., Pa.), F. Burgess and H. Salazar. *Cancer* 31(6):1433-1439, 1973.
- 4106 ULTRASTRUCTURE OF A VIRILIZING OVARIAN SERTOLI-LEYDIG CELL. TUMOR WITH FAMILIAL INCIDENCE. (E.) Murad, T. M. (Dept. Med., Ohio St. U., Columbus), R. Mancini and J. George. *Cancer* 31(6):1440-1450, 1973.
- 4107 ULTRASTRUCTURE OF BRONCHIAL ONCOCYTOMA. (E.) Fechner, R. E. (Baylor Coll. Med., Houston, Tex.) and B. R. Bentinck. *Cancer* 31(6):1451-1457, 1973.
- 4108 CUSHING'S SYNDROME AND BRONCHIAL CARCINOID TUMOR. (E.) Olurin, E. O. (U. Coll. Hosp., Ibadan, Nigeria), E. O. Sofowora, A. O. Afonja, T. M. Kolawole and T. A. Junaid. *Cancer* 31(6):1514-1519, 1973.
- 4109 OAT CELL CARCINOMA OF THE PANCREAS WITH ECTOPIC ACTH SECRETION. (E.) Corrin, B. (St. Thomas's Hosp., Med. Sch., London, England), E. D. Gilby, N. F. Jones and J. Patrick. *Cancer* 31(6):1523-1527, 1973.
- 4110 HODGKIN'S DISEASE IN TRUJILLO, PERU. CLINICAL AND HISTOLOGIC PRESENTATION. (E.) Albújar, P. F. (Belen Hosp., Trujillo, Peru). *Cancer* 31(6):1520-1522, 1973.
- 4111 HEMANGIOPERICYTOMA: ULTRASTRUCTURAL STUDY OF FIVE CASES. (E.) Battifora, H. (Northwestern U. Med. Sch., Chicago, Ill.). *Cancer* 31(6):1418-1432, 1973.
- 4112 DERMATOMYOSITIS AND CARCINOMA OF THE RECTUM (RESECTED). (E.) Navaratnam, A. (U. Hosp. Wales, Cardiff) and E. Waddington. *Br J Dermatol* 89(Suppl 9):43-46, 1973.
- 4113 PRIMARY MACROGLOBULINAEMIA AND LYMPHOMA. (E.) Navaratnam, A. (U. Hosp. Wales, Cardiff) and G. A. Hodgson. *Br J Dermatol* 89(Suppl 9):21-25, 1973.
- 4114 PITUITARY TUMORS IN WOMEN. (E.) Downing, T. A. (U. Colorado Sch. Med., Denver), T. Engel and G. Betz. *Obstet Gynecol* 42(2):182-185, 1973.
- 4115 STANDARDS FOR THE ASSESSMENT OF ESTROGEN RECEPTORS IN HUMAN BREAST CANCER. REPORT OF A WORKSHOP ON SEPTEMBER 29, 1972, AT THE ANTONI VAN LEEUWENHOEK-HUIS, AMSTERDAM. (E.) Heuson, J. C. (Chairman, E.O.R.T.C. Breast Cancer Cooperative Group, Bordet Inst., Brussels, Belgium). *Cancer* 9(5):379-381, 1973.
- 4116 THE MISSING Y CHROMOSOME AND HUMAN LEUKEMIA. (E.) Sandberg, A. A. (Roswell Park Mem. Inst., Buffalo, N.Y.) and M. Sakurai. *Lancet* (7799):375, 1973.
- 4117 PURIFICATION OF A POLY (ADP-RIBOSE) PROTEIN COMPLEX FROM EHRlich ASCITES TUMOR NUCLEI. (E.) Adamietz, P. (Eppendorf U. Hosp., Hamburg, West Germany) and H. Hilz. *Hoppe Seylers Z Physiol Chem* 353(6):845, 1972.
- 4118 NUCLEAR POLY(ADP-RIBOSYL)ATION DURING RESTRICTED MACROMOLECULAR SYNTHESIS OF HELA CELLS. (E.) Smulson, M. (Georgetown U., Sch. Med., Dentistry, Washington, D.C.). *Hoppe Seylers Z Physiol Chem* 353(6):849, 1972.
- 4119 PROPERTIES OF MAMMALIAN NUCLEAR AND MICROSOMAL NAD GLYCOHYDROLASES. (E.) Green, S. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.). *Hoppe Seylers Z Physiol Chem* 353(6):851, 1972.
- 4120 THYMOMA: FACTORS INFLUENCING PROGNOSIS. (E.) Bernatz, P. E. (Mayo Clin., Fdn., Rochester, Minn.), S. Khonsari, E. G. Harrison, Jr. and W. F. Taylor. *Surg Clin North Am* 53(4):885-892, 1973.
- 4121 RETROGRESSION OF FIBROADENOMAS OF THE BREAST. (E.) Kern, W. H. (Hosp. Good Samaritan Med. Ctr., Los Angeles, Calif.) and R. W. Clark. *Am J Surg* 126(1):59-62, 1973.
- 4122 PROLONGED SURVIVAL IN UNTREATED BREAST CANCER. (E.) Steckler, R. M. (U. Texas, M. D. Anderson Hosp., Tumor Inst., Houston) and R. G. Martin. *Am J Surg* 126(1):111-113, 1973.
- 4123 CYTOCHEMICAL STUDY OF NON-SPECIFIC ESTERASE, ACID AND ALKALINE PHOSPHATASE IN THE HUMAN THYROID NEOPLASMS. (E.) Smejkalova, E. (Res. Inst. Endocrinology, Prague, Czechoslovakia) and V. Smejkal. *Acta Histochem* 46(1):74-78, 1973.
- 4124 *IN VITRO* METHODS OF ASSESSING LYMPHOCYTE TRANSFORMATION IN PATIENTS UNDERGOING RADIOTHERAPY FOR BRONCHOGENIC CANCER. (E.) Jenkins, V. K. (U. Texas Med. Branch, Galveston), M. H. Olson and H. N. Ellis. *Tex Rep Biol Med* 31(1):19-28, 1973.

- 4125 CHIMERISM FOLLOWING FETAL TRANSFUSION. REPORT OF LEUCOCYTE HYBRIDIZATION AND INFANT WITH ACUTE LYMPHOCYTIC LEUKAEMIA. (E.) Turner, J. H. (Dept. Biostatistics, U. Pittsburgh, Pa.), D. L. Hutchinson and J. C. Petricciani. *Scand J Haematol* 10(5):358-366, 1973.
- 4126 DIFFERENTIATION OF LEUKAEMIC CELLS IN DIFFUSION CHAMBERS. A CYTOCHEMICAL AND MORPHOLOGICAL STUDY OF SHAY CHLOROLEUKAEMIA CELLS CULTIVATED IN DIFFUSION CHAMBERS *IN VIVO*. (E.) Vilpo, J. A. (2nd Dept. Path., U. Helsinki, Finland). *Scand J Haematol* 10(5):367-377, 1973.
- 4127 ALPHA₁-FETOPROTEIN CONTENT OF GASTRIC CARCINOMA AND HEPATIC METASTASES. (E.) Montplaisir, S. (Sch. Med., St. U. New York, Buffalo), B. Rabin, M. Pelletier, N. R. Rose and E. Alpert. *Dig Dis* 18(5):416-418, 1973.
- 4128 ESTROGEN EXCRETION WITH THE URINE IN PATIENTS WITH PRETUMOR MAMMARY GLAND DISEASES. (Rus.) Kozhnazarova, Ju. S. (Kazakh Res. Inst. Oncol., Radiol., Alma-Ata, USSR), N. N. Mezinova, M. G. Remizova and S. T. Iskhanova. *Vopr Onkol* 18(10):23-26, 1972.
- 4129 ADENOCARCINOMA AND CROHN'S DISEASE. A REPORT OF 2 CASES AND ANALYSIS OF THE LITERATURE. (E.) Darke, S. G. (London Hosp., England), A. G. Parks, J. L. Grogono and D. J. Pollock. *Br J Surg* 60(3):169-175, 1973.
- 4130 ULTRASTRUCTURAL STUDY OF TWO GONADOTROPHS IN RATS BEARING AN ADRENOCORTICAL CARCINOMA (SNELL 494). (E.) Nakayama, I. (Dept. Path., St. U. New York, Buffalo) and P. A. Nickerson. *Am J Pathol* 71(2):279-294, 1973.
- 4131 ALTERATIONS IN MITOCHONDRIAL ADENOSINE TRIPHOSPHATASE ACTIVITY RESULTING FROM MUTATION OF MITOCHONDRIAL DEOXYRIBONUCLEIC ACID. (E.) Shannon, C. (Dept. Biochem., Biophys., U. California, Davis), R. Enns, L. Wheelis, K. Burchiel and R. S. Criddle. *J Biol Chem* 248(9):3004-3011, 1973.
- 4132 CLAMS USED IN CANCER WORK. (E.) Anonymous. *Adhesives Age* 16(5):84, 1973.
- 4133 THE MODIFICATION OF DEOXYRIBONUCLEOHISTONE BY TRYPSIN AND CHYMOTRYPSIN. (E.) Chatterjee, S. (Oxford U., England) and I. O. Walker. *Eur J Biochem* 34(3):519-526, 1973.
- 4134 ANTICANCER DRUG DESIGN INVOLVING COMPLEXES OF AMINO-ACIDS AND METAL IONS. (E.) Williams, D. R. (U. St. Andrews, Scotland). *Inorg Chim Acta Rev* 6:123-133, 1972.
- 4135 CARBOCYCLIC ANALOG OF PURINE RIBONUCLEOSIDES WITH ANTILEUKEMIC ACTIVITY. (E.) Shealy, Y. F. (Kettering-Meyer Lab., Southern Res. Inst., Birmingham, Ala.) and J. D. Clayton. *J Pharm Sci* 62(5):858-859, 1973.
- 4136 IRREVERSIBLE INHIBITION OF RIBONUCLEOTIDE REDUCTASE FROM EHRlich TUMOR CELLS BY A MODULATOR ANALOG. (E.) Cory, J. G. (Coll. Med., U. South Florida, Tampa) and C. B. George. *Biochem Biophys Res Commun* 52(2):496-503, 1973.
- 4137 EFFECTS OF STEROIDS, FOLATE DEPRIVATION AND PROTEIN DEPRIVATION UPON TETRAHYDROFOLATE DEHYDROGENASE LEVELS IN MAMMALIAN TISSUES. (E.) Makulu, D. R. (Yale U. Sch. Med., New Haven, Conn.), E. F. Smith and J. R. Bertino. *Biochim Biophys Acta* 304(2):526-532, 1973.
- 4138 SOME REMARKS ABOUT NUCLEIC BASES. (E.) Sorarrain, O. M. (Natl. U. La Plata, Argentina) and E. A. Castro. *Chem Phys Lett* 19(3):422-426, 1973.
- 4139 THE EFFECT OF PHOTOSENSITIZING DYES AS ANTI-OXIDANTS ON THE AUTOXIDATION OF METHYL LINOLEATE AND THE SPECULATION ON THEIR UTILITY AS ANTICANCER AGENTS. (E.) Fukuzumi, K. (Nagoya U., Japan) and N. Ikeda. *J Sanyo Assoc Adv Sci Technol* 26(1):1-4, 1972.
- 4140 FIBRINOLYSIS AND CIRCULATING MALIGNANT CELLS. (E.) Salisbury, A. J. (Brompton Hosp., London, England), C. White, P. Tsolakidis, J. A. McKinna and J. D. Griffiths. *Surg Gynecol Obstet* 136(5):733-736, 1973.
- 4141 HISTOLOGY AND ULTRASTRUCTURE OF AN ONCOCYTIC ADENOMA OF THE HUMAN PITUITARY. (E.) Landolt, A. M. (Neurosurg. Clin., U. Zurich, Switzerland) and U. W. Oswald. *Cancer* 31(5):1099-1105, 1973.
- 4142 HODGKIN'S DISEASE OF THE ESOPHAGUS: SUCCESSFUL TREATMENT OF A RARE COMPLICATION. (E.) Morrison, F. S. (U. Mississippi Sch. Med., Jackson), F. Critz, W. T. Tatum and H. K. Stauss. *Cancer* 31(5):1244-1246, 1973.
- 4143 ANALOGIES BETWEEN EXPERIMENTAL AND HUMAN LYMPHOMA CUTIS. (E.) Maruyama, Y. (Dept. Radiation Med., U. Kentucky, Lexington), T. Mariani, E. P. Engels and R. A. Good. *Cancer* 31(5):1106-1113, 1973.
- 4144 DISSEMINATED INTRAVASCULAR COAGULATION IN CANCER PATIENTS: SUPPORTIVE EVIDENCE. (E.) Peck, S. D. (Presbyterian Med. Ctr., Denver, Colo.) and C. W. Reiquam. *Cancer* 31(5):1114-1119, 1973.

- 4145 FEATURES OF BENIGN BONE TUMORS OCCURRING IN CHILDHOOD. (Ger.) Lehner, M. (Zurich U. Child Dis. Clinic, Switzerland) and H. Gessendorfer. *Helv Chir Acta* 40(1-2):147-151, 1973.
- 4146 LEUKEMIA AND HEREDITY. (Ger.) Fekete, G. (Simmelweis Med. U., Budapest, Hungary), M. Dobos, D. Schuler and J. Fischer. *Acta Paediatr Acad Sci Hung* 13(4):387-393, 1972.
- 4147 GRANULOCYTIC DISEASES ACQUIRED IN LEUKEMIA AND UNMANAGEABLE ANEMIAS. (Fr.) Dreyfus, B. (No affiliation). *Nouv Rev Fr Hematol* 13(2):243-248, 1973.
- 4148 PORPHYRIA CUTANEA TARDA. REPORTING TWO CASES ASSOCIATED WITH CANCER OF THE LIVER. (Fr.) Rimbaud, P. (St. Charles Clin. Montpellier, France), J. Meynadier and J.-J. Guilhou. *Sem Hop Paris* 49(10):719-725, 1973.
- 4149 DERMATOGLYPHICS AND ACUTE LYMPHATIC LEUKEMIA IN CHILDREN. (It.) Colombo, A. (Pavia U. Pediat. Clin., Italy), M. C. Gasparoni, G. Biscatti and F. Severi. *Minerva Pediatr* 25(8):335-337, 1973.
- 4150 ISOLATION OF A HIGH MOLECULAR WEIGHT MESSENGER RNA FROM ANIMAL CELL POLYSOMES. (Fr.) Verger, C. (G. Roussy Inst., Villejuif, France). *Biochimie* 54(9):1147-1155, 1972.
- 4151 THE MIXED LYMPHOCYTIC CULTURE. POSSIBLE ROLE OF THE FIRST HL-A LOCUS REGION. (Fr.) Le Brun, A. (St. Louis Hosp. Blood Dis. Res. Inst., Paris, France), M. Sasportes and J. Dausset. *C R Acad Sci [D] (Paris)* 276(11):1763-1765, 1973.
- 4152 THE METABOLISM AND BINDING OF TESTOSTERONE IN ANDROGEN-DEPENDENT AND AUTONOMOUS TRANSPLANTABLE MOUSE MAMMARY TUMORS. (E.) Bruchovsky, N. (Dept. Med., U. Alberta, Edmonton, Canada) and J. W. Meakin. *Cancer Res* 33(7):1689-1695, 1973.
- 4153 ECTOPIC MENINGIOMAS. (E.) Shuangshoti, S. (Chulalongkorn U., Bangkok, Thailand) and R. Panyathanya. *Arch Otolaryngol* 98(2):102-105, 1973.
- 4154 IDIOSYNCRACIES OF PARATHYROID HYPERFUNCTION. (E.) Pollack, R. S. (U. California, Sch. Med., San Francisco). *Oncology* 27(5):394-405, 1973.
- 4155 KINETICS OF LYMPHOID CELLS IN TUMOR-BEARING MICE. (E.) Gillette, S. (Georgetown U. Sch. Med., Washington, D.C.) and J. A. Bellanti. *Cell Immunol* 8(2):311-320, 1973.
- 4156 CORRELATION OF PAPILLOEDEMA AND SELLA CHANGES WITH SITE OF TUMOUR (EXCLUDING PITUITARY TUMOURS). (E.) Cala, L. A. (C. Gairdner Hosp., Shenton Park, Australia). *Neuroradiology* 5(3):142-144, 1973.
- 4157 ISOZYME PATTERNS OF BRANCHED-CHAIN AMINO ACID TRANSAMINASE IN CULTURED RAT HEPATOCYTES. (E.) Ogawa, K. (Sch. Med., Tokushima U., Japan), A. Ichihara, H. Masuji and J. Sato. *Cancer Res* 33(3):449-453, 1973.
- 4158 SIGNIFICANCE OF POSTOPERATIVE ESTROGEN THERAPY ON THE OCCURRENCE AND CLINICAL COURSE OF CANCER. (E.) Byrd, B. F., Jr. (St. Thomas Hosp., Nashville, Tenn.), J. C. Burch and W. K. Vaughn. *Ann Surg* 177(5):626-631, 1973.
- 4159 THE TOTAL N-ACETYL NEURAMINIC ACID CONTENT OF HUMAN NORMAL AND LYMPHATIC LEUKAEMIC LYMPHOCYTES. (E.) McClelland, D. A. (Royal Victoria Hosp., Belfast, Northern Ireland) and J. M. Bridges. *Br J Cancer* 27(2):114-119, 1973.
- 4160 KINETIC DIFFERENCES OF AUTOTRANSFUSED ³H-CYTIDINE LABELED BLOOD LYMPHOCYTES IN LEUKEMIC AND NON-LEUKEMIC LYMPHOMA PATIENTS. (E.) Bremer, K. (Ctr. Basic Clin. Res., U. Ulm, West Germany), T. M. Fliedner and P. Schick. *Eur J Cancer* 9(2):113-124, 1973.
- 4161 MELANOMAS OF VULVA AND VAGINA. (E.) Fenn, M. E. (U. Michigan Med. Ctr., Ann Arbor) and M. R. Abell. *Obstet Gynecol* 41(6):902-911, 1973.
- 4162 SERUM PROTEINS IN PROSTATIC CANCER. I. RELATIONSHIP BETWEEN CLINICAL STAGE AND LEVEL. (E.) Ablin, R. J. (Southern Illinois U. Sch. Med., Springfield), H. J. Gonder and W. A. Soanes. *J Urol* 110(2):238-241, 1973.
- 4163 CENTRAL NERVOUS SYSTEM LESIONS IN CHILDHOOD LEUKAMIA. (E.) Robson, G. S. (Royal Children's Hosp., Melbourne, Australia) and R. McD. Anderson. *Med J Aust* 1(23):1134-1137, 1973.
- 4164 DEVELOPMENT OF THE VASCULAR SYSTEM IN THE HAMSTER MALIGNANT NEURILEMMOMA. (E.) Eddy, H. A. (U. Rochester Clinical Radiation Res. Ctr., Rochester, N.Y.) and G. W. Casarett. *Microvasc Res* 6(1):63-82, 1973.
- 4165 LONG-TIME OBSERVATIONS ON THE BLALOCK-TAUSSIG OPERATION. V. NEOPLASMS IN TETRALOGY OF FALLOT. (E.) Mulvihill, J. J. (Natl. Cancer Inst., Bethesda, Md.), R. W. Miller and H. B. Taussig. *Johns Hopkins Med J* 133(1):16-18, 1973.

- 4166 TUMOURS OF THE GLOMUS JUGULARE. (E.) Koelmeyer, T. D. (Middlemore Hosp., Auckland, New Zealand). *Med J Aust* 1(18):895-897, 1973.
- 4167 NECROTIC INTRADUCT BREAST CARCINOMAS SIMULATING INFLAMMATORY LESIONS. (E.) Jones, E. L. (Gen. Hosp., Birmingham, England), B. W. Godling and G. D. Oates. *J Pathol* 110(1):101-104, 1973.
- 4168 PIGMENTED PROGNOMA IN A DERMOID CYST OF THE OVARY. (E.) Sinniah, R. (Queen's Hosp., Belfast, Ireland) and F. V. O'Brien. *J Pathol* 109(4):357-359, 1973.
- 4169 THE NATURE OF THE MUTATION IN FAMILIAL MULTIPLE POLYPOSIS: PAPILLARY CARCINOMA OF THE THYROID, BRAIN TUMORS AND FAMILIAL MULTIPLE POLYPOSIS. (E.) Smith, W. G. (El Paso, Texas) and B. Kern. *Dis Colon Rectum* 16(4):264-271, 1973.
- 4170 HODGKIN'S DISEASE OF THE MANDIBLE: REPORT OF CASE. (E.) Stern, N. S. (Montefiore Hosp., Pittsburgh, Pa.) and D. R. Shensa. *J Oral Surg* 31(8):628-631, 1973.
- 4171 ADRENOCORTICAL CARCINOMA WITH AN ISOLATED MINERALOCORTICOID EXCESS AND RECURRENCE FOURTEEN YEARS AFTER REMOVAL OF THE TUMOR. (E.) Miyazaki, G. (Mishima Social Insurance Hosp., Japan), T. Sasano, T. Torikai and S. Fukuchi. *Tohoku J Exp Med* 109(4):365-375, 1973.
- 4172 IN VITRO STEROL AND STEROID BIOGENESIS BY A FEMINIZING ADRENOCORTICAL CARCINOMA. (E.) Mathur, R. S. (Med. U. South Carolina, Charleston), L. O. Williamson, L. O. Moody and E. Diczfalussy. *Acta Endocrinol* 73(3):518-530, 1973.
- 4173 HYPOBILIRUBINEMIA ASSOCIATED WITH HEMANGIOMA OF THE LIVER. (E.) Martinez, J. (Dept. Med., Thomas Jefferson U., Philadelphia, Pa.), S. Shapiro, R. R. Holburn and R. A. Carabasi. *Am Clin Pathol* 60(2):192-197, 1973.
- 4174 PULMONARY LYMPHANGIOMYOMA WITH RENAL ANGIOLIPOMAS. (E.) Leeds, S. E. (Mt. Zion Hosp. Med. Ctr., San Francisco), M. A. Benioff and Ortega. *Calif Med* 119(2):74-78, 1973.
- 4175 ADENOCARCINOMA OF THE LARYNX. (E.) Dogra, T. S. (Wythenshawe Hosp., Manchester, England). *J Laryngol Otol* 87(8):685-689, 1973.
- 4176 TUMORS OF THE TUNICA VAGINALIS. (E.) Remzi, D. (Hacettepe U. Sch. Med., Ankara, Turkey). *South Med J* 66(7):841-842, 1973.
- 4177 LYMPHOGRAPHIC FINDINGS IN A SERIES OF 258 PATIENTS WITH TUMORS OF THE TESTES. (E.) de Roo, T. (Central Hosp., Alkmaar Eindhoven, Netherlands) and S. H. van Minden. *Lymphology* 6(2):97-100, 1973.
- 4178 MULTIFOCAL GRANULAR CELL MYOBLASTOMA. REPORT OF A CASE INVOLVING TRACHEA, STOMACH AND ANTERIOR ABDOMINAL WALL. (E.) Krouse, T. B. (Med. Coll. Pennsylvania, Philadelphia) and J. Mobini. *Arch Pathol* 96(2):95-99, 1973.
- 4179 PEDIATRIC AND ADULT PHEOCHROMOCYTOMAS. A BIOCHEMICAL AND CLINICAL COMPARISON. (E.) Freier, D. T. (U. Michigan Med. Ctr., Ann Arbor), E. S. Tank and T. S. Harrison. *Arch Surg* 107(2):252-255, 1973.
- 4180 "DENUDING CYSTITIS" AND IN SITU UROTHELIAL CARCINOMA. (E.) Elliott, G. B. (Vancouver General Hosp., British Columbia, Canada), P. J. Moloney and G. H. Anderson. *Arch Pathol* 96(2):91-94, 1973.
- 4181 PERIPITUITARY GLAND INVOLVEMENT IN ACUTE LEUKEMIA IN ADULTS. (E.) Masse, S. R. (U. Texas M.D. Anderson Hosp., Houston), R. W. Wolk and R. H. Conklin. *Arch Pathol* 96(2):141-142, 1973.
- 4182 NONPARATHYROID HUMORAL HYPERCALCEMIA IN PATIENTS WITH NEOPLASTIC DISEASES. (E.) Powell, D. (Harvard Med. Sch., Boston, Mass.), F. R. Singer, T. M. Murray, C. Minkin and J. T. Potts, Jr. *N Engl J Med* 289(4):176-181, 1973.
- 4183 PRODUCTION OF HISTAMINE-LIKE AND PROSTAGLANDIN-LIKE SUBSTANCES FROM SERUM INCUBATED WITH RAT, DOG, MOUSE OR HUMAN TUMOURS. (E.) Apps, M. C. P. (Dept. Path., U. Cambridge, England) and D. B. Cater. *Br J Exp Pathol* 54(2):203-221, 1973.
- 4184 EARLY CONTACTS BETWEEN NORMAL FIBROBLASTS AND MOUSE SARCOMA CELLS. AN ULTRASTRUCTURAL STUDY. (E.) Heaysman, J. E. M. (Dept. Zoology, U. Coll. London, England) and S. M. Pegrum. *Exp Cell Res* 78(2):479-480, 1973.
- 4185 CELL PROLIFERATION IN THE CASTRATE MOUSE SEMINAL VESICLE IN RESPONSE TO TESTOSTERONE PROPIONATE. I. EXPERIMENTAL OBSERVATIONS. (E.) Morley, A. R. (Dept. Med. Statistics, Path., U. Newcastle upon Tyne, England), N. A. Wright and D. Appleton. *Cell Tissue Kinet* 6(3):239-246, 1973.
- 4186 CONTROL OF EPITHELIAL CELL PROLIFERATION IN THE SMALL INTESTINAL CRYPT. (E.) Tutton, P. J. M. (Dept. Anat., Monash U., Victoria, Australia). *Cell Tissue Kinet* 6(3):211-216, 1973.

- 4187 THE EVOLUTION OF NUMBER OF BONE MARROW STEM CELLS IN MICE WITH L₁₂₁₀ LEUKEMIA. (E.) Chevalier, C. (Inst. Radiobiol. Clin, INSERM, Villejuif, France) and E. Frindel. *Biomedicine* 19(4):177-179, 1973.
- 4188 REVIEW OF CARCINOMA OF PENIS AT MULAGO. (E.) Bhana, D. (Makerere Med. Sch., Kampala, Uganda) and S. K. Kyalwazi. *East Afr Med J* 49(12):996-1001, 1972.
- 4189 CLONAL EVOLUTION DURING ACUTE LEUKEMIA IN A MONGOL CHILD. (Fr.) Berger, R. (No affiliation), C. Weisgerber and J. Bernard. *Nouv Rev Fr Hematol* 13(2):229-236, 1973.
- 4190 SPONTANEOUS CYCLIC LEUKOCYTOSIS IN A CASE OF CHRONIC MYELOID LEUKEMIA. (Fr.) Delobel, J. (No affiliation), P. Charbord, Ph. Passa and J. Bernard. *Nouv Rev Fr Hematol* 13(2):221-228, 1973.
- 4191 ULTRASTRUCTURE AND PHYSICO-CHEMICAL PROPERTIES OF GLYCOGEN FROM ASCITES HEPATOMA AHT3. (E.) Iwamasa, T. (Kumamoto U. Sch. Med., Japan). *Acta Histochem Cytochem* 5(3):117-124, 1972.
- 4192 DIFFERENTIATION AND CELL GROWTH BY SYMMETRICAL AND ASYMMETRICAL MITOSIS: A HYPOTHESIS. (E.) Heyden, H. W. v. (Med. U. Clin. II, Tübingen, West Germany) and D. v. Heyden. *Perspect Biol Med* 16(3):349-356, 1973.
- 4193 SOME DETERMINANTS OF DRUG RESPONSIVENESS IN BOVINE LEUKEMIA CELLS. (E.) Kessel, D. (U. Rochester Sch. Med., Dentistry, New York), D. C. Dodd and T. C. Hall. *Biochem Pharmacol* 22(10):1161-1164, 1973.
- 4194 TISSUE CULTURE, ELECTRON MICROSCOPIC AND ENZYME HISTOCHEMICAL INVESTIGATIONS ON A SYMPATHETIC GANGLIONEUROBLASTOMA. (E.) Gullotta, F. (Inst. Neuropath., U. Bonn, West Germany), E. Fliedner, R. Wüllenweber and G. Orf. *Acta Neuropathol (Berl)* 24(2):107-116, 1973.
- 4195 EVALUATION OF THE TUMORIGENIC POTENTIAL OF VERMICULITE BY INTRAPLEURAL INJECTION IN RATS. (E.) Hunter, B. (Huntingdon Res. Ctr., England) and C. Thomson. *Br J Ind Med* 30(2):167-173, 1973.
- 4196 THE NATURE OF THE TRANSFORMING LYMPHOCYTE IN CHRONIC LYMPHOCYTIC LEUKEMIA. (E.) Schweitzer, M. (Central Lab. Netherlands Red Cross Blood Transfusion Service, Amsterdam), C. J. M. Melief and V. P. Eijssvoegel. *Eur J Immunol* 3(3):121-126, 1973.
- 4197 THE FIBRINOLYTIC ENZYME SYSTEM IN MALIGNANT LYMPHOMAS. (E.) Ogston, D. (Dept. Med., Path., U. Aberdeen, Scotland) and A. A. Dawson. *Acta Haematol (Basel)* 49(2):89-95, 1973.
- 4198 ROLE OF THE TUMOR PHOSPHOLIPIDS IN THE ACCUMULATION OF ⁶⁷GA-CITRATE. (E.) Anghileri, L. J. (Ruhr-U. Clin., Essen, West Germany). *J Nucl Biol Med* 16(1):21-23, 1972.
- 4199 HISTOCHEMICAL STUDY OF MUCOPOLYSACCHARIDE IN GASTRIC CANCER. (E.) Hirose, S. (Osaka U. Med. Sch., Japan), M. Yasutomi, N. Murai, Z. Iwasa, M. Hirose, K. Shindo, R. Aso, N. Kikkawa and A. K. A. Razzaq. *Acta Histochem Cytochem* 5(3):153-160, 1972.
- 4200 EFFECTS OF METABOLIC INHIBITORS ON GIANT CELL FORMATION IN *OEDOGONIUM CARDIACUM*. (E.) Banerjee, S. N. (Hamilton Clin., Ontario, Canada) and R. J. Horsley. *Radiat Res* 54(1):121-129, 1973.

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CARCINOGENESIS ABSTRACTS

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PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

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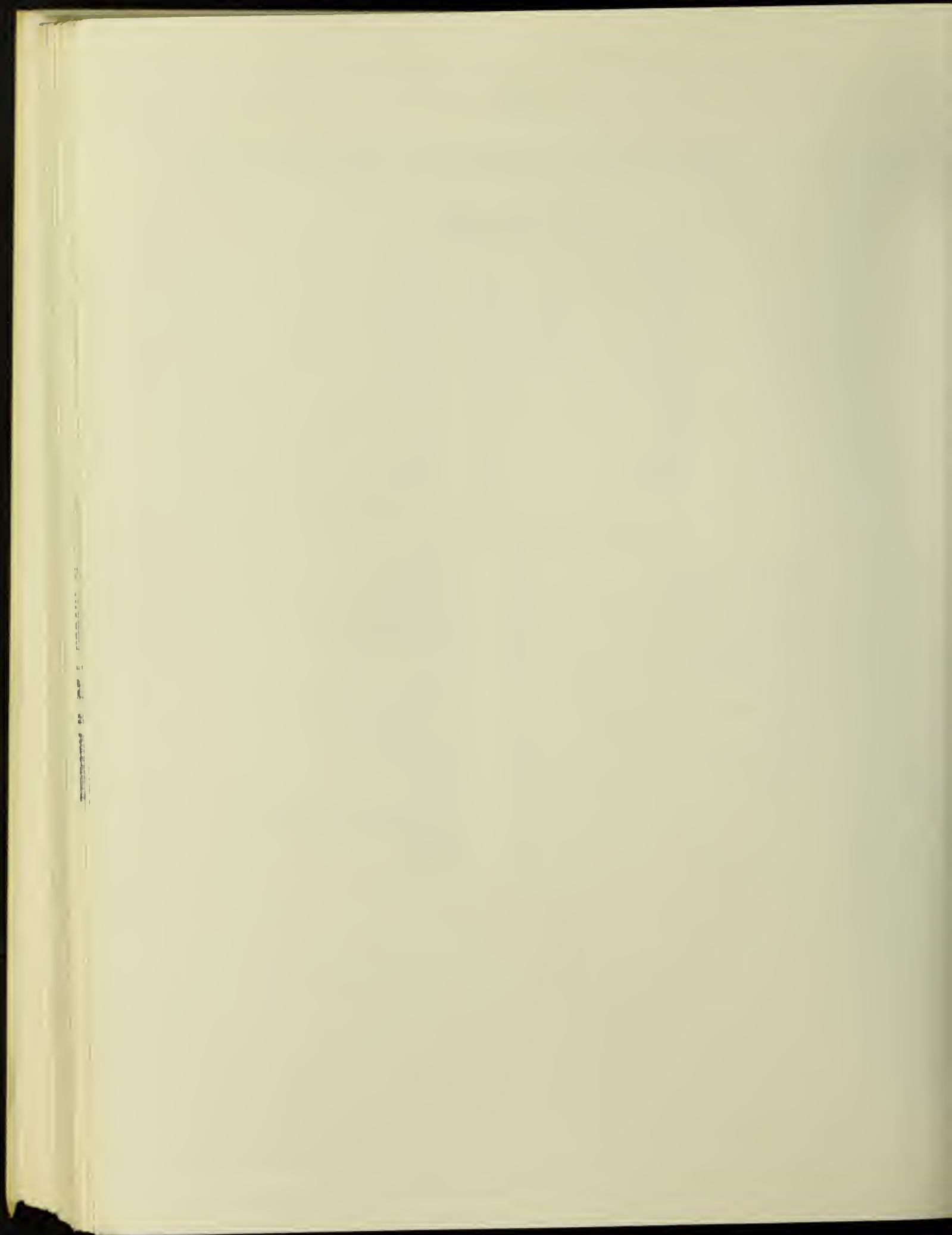
Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
Ind.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotrophic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
µg	microgram(s)	resp.	respectively
hr	hour(s)	RNA	ribonucleic acid
i.m.	intramuscular	s.c.	subcutaneous
i.p.	intraperitoneal	sec	second(s)
IU	international unit(s)	U	unit(s)
i.v.	intravenous	UV	ultraviolet
kg	kilogram(s)	WBC	white blood cells (leukocytes), white blood count
LD ₅₀	median lethal dose(s)	wk	week(s)
m	meter(s)	wt	weight(s)
M	molar	yr	year(s)
mEq	milliequivalent(s)		
mM	millimolar		
µM	micromolar		
mC, µC	milli-,microcurie(s)		



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- 4201 MICROBIOLOGY: HAZARDOUS PROFESSION FACES NEW UNCERTAINTIES. (E.) Wade, N. (Cold Springs Harbor Lab., N.Y.). *Science* 182(4112):566-567, 1973.

Since the turn of the century, some 3500 cases of laboratory-acquired infections have been reported, more than 150 of which resulted in death. The risks are, if anything, increasing as work with viruses is expanded and includes viruses suspected of causing cancer in man, even under the most stringent safety conditions that can be devised, such as those at the former biological warfare laboratories at Fort Detrick, Maryland. For example, self-inoculation with a syringe and inhalation can lead to infection. The trend is to use viruses in more highly concentrated forms and solutions now in laboratory use contain 100 to 1000 times more virus than a few years ago. There are also dangers posed by the new kinds of virus that can be created in the laboratory and which, if they escaped, might constitute a threat to public health. One possible experiment calls for a virus, which is a combination of the DNA of SV40 and certain bacterial genes, to be made to infect *E. coli*. Should SV40 infected *E. coli* escape from the laboratory it might become established in the population at large. Hybrids between SV40 and some of the human adenoviruses could become established in the tonsils of young children and become part of the human experience for generations to come should they escape from the laboratory. Despite the great danger, safety practices vary widely from one laboratory to another. (No references)

- 4202 THE HEALTH HAZARDS OF PLASTICS. (E.) Eckardt, R. E. (Med. Res. Div., Esso Res. Engineering Co., Linden, N.J.) and R. Hindin. *J Occup Med* 15(10):808-819, 1973.

The toxicology of plastics is reviewed, including the direct toxic effects, dermatological problems, the association of acro-osteolysis with manufacturing polyvinyl chloride, hazards from plastic fillers and the thermal decomposition of plastics, the potential hazards from the medical use of plastics and the association between plastics and carcinogenicity. One theory regarding the latter association is that carcinogenesis is dependent on the physical properties of plastics. This theory is supported by the observation that the porosity of the polymer is significant in the tumorigenic effect of implanted film in laboratory animals. A significantly higher incidence of sarcomas formed in mice that had received polyethylene discs having a smooth surface than discs that had a roughened surface. Another theory suggests that a chemical interaction between the implanted polymer and adjacent tissue leads to tumorigenesis. Using radioactively tagged material, it was shown that polymers were in fact degraded and metabolized after implantation in rats, although the amount of breakdown was minute and no metabolites were identified. Polymer tumorigenesis is regarded by some researchers as an evolutionary process. This concept is supported by several findings of studies on the capsule which develops around polymer implants. Tumors induced in mice by s.c. implantation of plastic films include four histologi-

cal classes: well differentiated fibrosarcomas, spindle-cell sarcomas, anaplastic round-cell sarcomas without giant cells and anaplastic sarcomas with giant cells. The implications of polymer tumorigenesis are significant from a medical standpoint, especially regarding prostheses, breast implants and intrauterine contraceptive devices (IUD's). The three types of plastic presently used for IUD's induced fibromas or fibrosarcomas when implanted s.c. in rats. A study of the endometrial changes in 208 women using polyethylene IUD's is described. Of the 107 symptomatic patients (bleeding, pelvic pain, and vaginal discharge) 25.2% showed significant lesions, including diffuse inflammatory changes and atypical glandular hyperplasia with quasi-neoplastic architecture. Minor lesions, including a lymphocytosis, minute foci of granulation tissue, focal disappearance of glands, and endometrial morphology asynchronous by seven days or more from the stated day of the cycle, were seen in 45.8% of the symptomatic patients. Of the asymptomatic patients, 9.7% showed significant lesions and 50.5% showed minor changes. (149 references)

- 4203 AN OVERVIEW OF TRANSPLENTAL CHEMICAL CARCINOGENESIS. (E.) Rice, J. M. (Nat'l. Cancer Inst., Bethesda, Md.). *Teratology* 8(2):113-125, 1973.

Transplacental carcinogenesis is surveyed with emphasis on major problems currently constituting active areas of research. These include identification of transplacental chemical carcinogens and the tissues they preferentially affect in different species; enzyme-mediated metabolism of carcinogens; formation of reactive metabolites in maternal, placental, and fetal tissues; transplacental passage of carcinogens and their localization in fetal tissues; changing susceptibilities of different tissues during embryonic and fetal development; differences in susceptibility to a given agent between corresponding tissues of fetus and adult; carcinogenic effects in later generations not directly exposed to carcinogens *in utero*; tissue culture systems for identification of transformed cells used in combination with transplacental exposure to carcinogens; and relevance of animal experiments to cancer in man, including the special case of cancer in childhood and the possible interaction of teratogenic and carcinogenic agents. It is noted that both retinoblastoma and Wilms tumor in man may be inducible by a combination of teratogenic and transplacental carcinogenic effects in genetically susceptible individuals. Another childhood neoplasm that may possibly be induced by transplacental carcinogens is neuroblastoma. The lack of exact animal models for these tumors prevents investigation of regulatory factors involved in their behavior. (89 references)

- 4204 BENEFITS OF INTEGRATION. (E.) Lewin, R. (No affiliation). *New Scientist* 60(87):401-403, 1973.

Vertical transmission of viral genetic material (passage from parent to offspring) can be achieved

in a number of ways, including genetic trans-mission (intimate association of the virus with the genetic material of the germ cells). Many viruses achieve this intimacy by physically integrating their genomes into the hosts', seemingly preferably in the protein coding regions of host DNA chains. The enzyme reverse transcriptase, or RNA-direct DNA polymerase, is used by the RNA virus to produce a DNA copy of itself (provirus) prior to integrating with the host. The virus then appears to slip into and out of the host's genes quite frequently, often removing a piece of the host cell's DNA or leaving a piece of its own DNA as it is excised. It appears that a similar process may occur within normal cells. Reverse transcriptase, which has been discovered in a number of embryonic tissues, may play a role in cell differentiation. According to this theory, (provirus theory) the information coded for in genes might be shifted around within and between cells via RNA intermediates, thus exploiting the variability inherent in the genetic information. However, since this process is not always precise, wholly new DNA sequences could be created within the lifetime of a single organism simply from the mistakes of the provirus system. Some of the mistakes could produce new DNA inimical to the cell; thus the DNA sequences necessary for cancer formation might easily be assembled, as might an RNA virus. (No references)

- 4205 MOLECULAR BASIS FOR VIRUS-INDUCED TUMORS.
(Ger.) Chandra, P. (Inst. Ther. Biochem. U. Frankfurt/Main, Germany), D. Gericke, F. Zunino and R. Thorbeck. *Klin Wochenschr* 51(16):781-790, 1973.

After a brief review of the classification and properties of oncogenic DNA and RNA viruses, the role of these viruses in cell transformation and oncogenesis is considered. By using reverse transcriptase inhibitors, a correlation has been established between reverse transcriptase activity and the infectivity and oncogenicity of RNA viruses. Structure-activity relationships are described for distamycin A and three analogs with different numbers of pyrrole radicals in the molecule and for daunomycin and four of its derivatives (adriamycin, 13-dihydrodaunomycin, N-guanidinacetamide daunomycin, and N-acetyldaunomycin). The presence of particles resembling RNA viruses in human tumor cells has been explained by oncogene and provirus theories. According to the former, only a part of these virogenes, the oncogene, is responsible for neoplastic transformation which usually occurs in undifferentiated proliferating cells such as those found in the early embryo. This virogene, which is normally inactive, can be activated by carcinogens, radiation, or other oncogenic DNA or RNA viruses. Activation results in derepression of information normally controlled by a repressor. Virus replication, malignant tumor development or both occur, depending upon a number of other factors. According to the provirus hypothesis, C-particles originate from genes, some of which contain provirus, a product of cellular RNA template with reverse transcriptase. Tumors are pro-

duced when these proviruses are changed or abnormally integrated. Although these theories are not incompatible, no definite proof exists for the validity of either. Neither theory accounts for tumor production by changes in DNA or explains how carcinogens induce tumors. (38 references)

- 4206 CURRENT ASPECTS OF TUMOR IMMUNOLOGY. (E.) Weiss, D. W. (Jerusalem, Israel). *Isr J Med Sci* 9(3):205-216, 1973.

Reports of original research as well as discursive articles on a number of problems which are central in the study of the immunological parameters of host-tumor associations are brought together in this review. Topics presented include: changes in cell surface characteristics which occur during neoplastic transformation or during the subsequent development of the transformed clones, their significance for host immunological responsiveness and the immunochemical analysis of the surface of transformed cells; detailed histological evidence for immunological intervention by the human host in the course of preneoplastic and neoplastic changes of the breast; cellular aspects of the immune response to tumor cells *in vitro* and *in vivo*, and the problems of blocking antibody, excessive amounts of tumor antigen and specific desensitization; development of animal models of possible immunotherapeutic manipulations in man; specific and non-specific immunological aspects of a recently developed model for tumor immunology - carcinogen-induced hepatomas of guinea pigs; incidental destruction of tumors by immune responses directed at unrelated antigens in the vicinity; and altered transplantation characteristics of seemingly normal tissues of hosts bearing neoplastic cells or oncogenic viral agents. (53 references)

- 4207 LOSS OF SUPPRESSOR FUNCTION AS A CAUSE OF LYMPHOID MALIGNANCY. (E.) Gershwin, M. E. (Natl. Inst. Arthritis, Metabolism, Digestive Diseases, Bethesda, Md.) and A. D. Steinberg. *Lancet* (7839):1174-1176, 1973.

The increased frequency of lymphoreticular malignancy in relation to other neoplasms is discussed. One explanation for this phenomenon holds that the high incidence of lymphoma in immunological deficiency is due to depressed surveillance on the part of the immunological system. Although this theory may be correct for nonlymphoid tumors, the development of lymphoreticular malignancies seems to be due to another defect. Different lymphoid-cell populations have been shown to interact in various immunological responses, including the suppression of humoral and cellular immunity. Additional data indicate the existence of thymic regulation by suppression of the antibody response to several antigens which require little thymic helper function; suppression has also been demonstrated for antigens requiring thymic helper cells for a maximum antibody response. It is suggested that the underlying defect in many diseases which predispose the patient to lymphoreticular malignancy is a loss of thymic suppression. Without adequate suppression,

rapid proliferation of lymphoreticular cells would increase the probability of the emergence of a malignant clone. Continued rapid proliferation of such a clone would result in its becoming a dominant cell population, leading to overt disease. The reason for the loss of suppressor cells is unclear and may represent aging, genetic, viral, or chemotherapeutic influence. This hypothesis has implications for prevention of the loss of suppressor function or the restoration of suppressor function to prevent or retard malignant change. (29 references)

- 4208 LONGEVITY IN RADIATED HUMAN POPULATIONS, WITH PARTICULAR REFERENCE TO THE ATOMIC BOMB SURVIVORS. (E.) Anderson, R. E. (Univ. New Mexico Sch. Med., Albuquerque). *Am J Med* 55(5):643-656, 1973.

Life shortening is one of the late consequences of exposure of a variety of laboratory animals to biologically significant amounts of ionizing radiation. Several human populations accidentally and/or therapeutically exposed to radiation were studied and life shortening does appear among the radium dial painters and pioneer American radiologists even when appropriate corrections are made for the known tumorigenic effects of such exposure. Life shortening is at most equivocally reflected in the data available concerning the survivors of the atomic bombs of Hiroshima and Nagasaki. Data concerning children irradiated for enlarged thymus and other benign lesions of the neck and chest indicate that the life shortening which occurs is due primarily to the development of leukemia. In children irradiated *in utero* during diagnostic pelvimetry, information is not yet sufficiently available to compute any effect this irradiation had on the life span of the child. (58 references)

- 4209 CANCER OF THE KIDNEY--ETIOLOGY, EPIDEMIOLOGY, AND PATHOLOGY. (E.) Bennington, J. L. (Dept. Path., Children's Hosp. San Francisco, Calif.). *Cancer* 32(5):1017-1029, 1973.

Renal adenocarcinoma represents approximately 1% of all malignancies exclusive of skin cancer. It is extremely rare in children and young adults and its incidence is over three times as great in men as in women. Familial occurrence is rare, although there is a high frequency of occurrence in patients with Lindau's disease. Naturally occurring renal adenocarcinoma is uncommon in lower animals except in rare instances of familial tumors in rodents. However, in laboratory animals, renal adenocarcinomas are readily induced by a variety of carcinogens including chemical, physical, and viral agents. The implications of these studies to the epidemiology of human renal adenocarcinoma are discussed. Renal adenocarcinomas arise from the proximal convoluted tubule and are remarkably similar histologically and electronmicroscopically. Typically, these tumors are composed of cells which are clear or granular, a function of the cellular content of glycogen, lipids, and cytoplasmic organelles-- and may be arranged in solid, cystic, trabecular, tubular,

or papillary patterns. About 1.8% of all renal adenocarcinomas are sarcomatoid mimicking fibrosarcoma, rhabdomyosarcoma, or liposarcoma. These various histologic patterns are poorly related to survival, except for the sarcomatoid tumors which are regarded as highly aggressive. Renal cortical parenchymal tumors resembling the renal adenocarcinoma but less than 3 cm in diameter have been regarded as adenomas because of their low frequency of metastases. Based on the many points of similarity between the renal adenocarcinoma and the renal adenoma, it is postulated that these tumors are small renal adenocarcinomas which have not yet grown large enough to metastasize. (40 references)

- 4210 IS CANCER CAUSED BY CHANGES IN THE CELL SURFACE? (E.) Schnebli, H. P. (Friedrich Miescher Inst., Basel, Switzerland) and M. M. Burger. *Umschau* 73(16):487-490, 1973.

In a review, primarily of their own work, the authors describe experiments performed with cultures of normal 3T3 mouse fibroblasts and fibroblasts transformed by mouse polyoma virus or SV 40. Mild trypsinization of normal fibroblasts caused surface changes similar to those in transformed fibroblasts. Trypsin exposed 2 antigens (Forssman antigen and S-antigen), and lectins agglutinated cells. This effect was reversible, and surface changes were repaired in 2-6 hr. Under favorable conditions trypsin also caused mitosis in 80% of the cells in a monolayer and temporarily eliminated contact inhibition. Monovalent nonagglutinating concanavalin A produced contact inhibition in transformed cells; removal of concanavalin A from the cells eliminated it. Previous studies have shown that protease activity on the surface of transformed cells is higher than that on normal cells. Five different protease inhibitors, belonging to 3 different chemical classes of compounds, inhibited growth of transformed cells but did not completely restore contact inhibition. Research should be done to determine whether differences between the surfaces of normal and transformed cells are caused by differences in chemical composition or by differences in the topological distribution of carbohydrate-containing components. The results might help to explain contact inhibition, tumor immunology, tumor invasion, and the decreased adhesion of tumor cells. (14 references)

- 4211 BRONCHIOLAR CARCINOMA: A REVIEW OF 152 PATIENTS. (E.) Martini, N. (Memorial Sloan-Kettering Cancer Ctr., New York, N.Y.), M. M. Khafagy, M. M. Melamed, R. B. Golbey and E. J. Beattie, Jr. *Clin Bull Sloan-Kettering Cancer Ctr* 3(3):98-101, 1973.

The records of 152 patients treated for bronchiolar carcinoma from 1961 through 1968 at the Memorial Sloan-Kettering Cancer Center were reviewed. These cases represented 9% of all cases of primary lung cancer treated during this period. There was a 2:1 predominance of males with an overall median age of 57 yr. Only 7% had never smoked. Thirty-two percent of the patients had no symptoms, their lesions being detected by routine x-ray as either pulmonary mass or infiltrate or pleural

effusion. Cough, dyspnea, and hemoptysis were most common in those with symptoms. Diagnosis was initially established by sputum cytology, pleural cytology, or mass biopsy in three-fifths and by thoracotomy in two-fifths of the patients. One-half had pleural extension or distant metastases at the time of diagnosis. Treatment included resection in 68 patients with or without supplemental irradiation, external irradiation alone in 25, irradiation plus chemotherapy in 18, or chemotherapy alone in 22. Of all patients in this study, 28 (18%) were alive at three yr, 17 (11%) were alive at five yr, and 10 (7%) were alive after five to nine yr. All long-term survivors had resectable lesions and were treated by surgery alone. Prognosis was best in those with localized, asymptomatic disease at the time of the diagnosis. (14 references)

- 4212 ANGIOSARCOMA OF THE HEART--REPORT OF A
 CASE AND REVIEW OF THE LITERATURE. (E.)
Ohtsuki, Y. (Okayama U. Med. Sch., Japan), S. Kobayashi, T. Hayashi and M. Ohmori. *Acta Pathol Jap* 23(2):407-413, 1973.

Autopsy of a 69-yr-old woman who had had clinical signs of congestive heart failure and who had not responded to therapy revealed the presence of an angiosarcoma of the right atrial wall with hemopericardium, cardiomegaly, and pleural effusion. Histologically, the tumor consisted of spindle-shaped cells with pale eosinophilic cytoplasm which formed sinusoidal spaces. Growth was invasive with myometrial hemorrhage and necrosis. Tumor cells were moderately atypical with sparsely scattered giant cells and mitotic figures. No metastatic disease was found. Electron microscopic study showed tumor cells with irregularly shaped nuclei and scant cytoplasm which contained a small, cystic granular endoplasmic reticulum, a few oval mitochondria and randomly oriented filaments. Review of the literature showed that only 46 cases of angiosarcoma of the heart have been reported, with a 2:1 predominance of males and a median age of 40. Sixty-three percent of these tumors were located in the right atrium. Sixty percent metastasized to the lung, with liver and regional nodes being the next two most frequent sites. The disease ran a rapid clinical course often lasting less than nine months. (18 references)

- 4213 TUMOR BIOLOGY, PRECANCEROUS STATES, CAN-
 CER PREVENTION. (Ger.) Tanneberger, S.
(Ctr. Inst. Cancer Res., Berlin-Buch, Germany).
Arch Geschwulstforsch 41(3):221-227, 1973.

Although many genetic and epigenetic mechanisms have been proposed to account for malignant transformation, more is known about sites which carcinogens attack in the cell. Some evidence that implicates genetic instability, aging, hormone imbalances, viruses, and chemical carcinogens in the development of cancer is briefly considered. If immunological defense mechanisms which partially or completely eliminate neoplastic cells are not operative, cancer develops in patients with precancerous states, such as xeroderma pigmentosum and immune deficiency diseases, and in patients being treated with immunosuppressants. In some cases

cancer can be prevented by identifying high-risk individuals by cytological methods, karyotyping, cytophotometric DNA determinations, establishment of enzyme distribution patterns which can be correlated with cell kinetics, and immunological parameters such as carcinoembryonic antigen. However, no one method suffices for biological characterization of human tumors. (40 references)

- 4214 ETIOLOGY, EPIDEMIOLOGY, AND PATHOLOGY OF
 PROSTATIC CANCER. (E.) Franks, L. M.
(Dept. Cellular Path., Imperial Cancer Res. Fund,
London, England). *Cancer* 32(5):1092-1095, 1973.

Hormones appear to stimulate the development and maintenance of the prostatic epithelium so that a sufficient number of cells is present in which malignant change can occur. Pituitary hormones may be involved in the etiology of prostatic cancer, but it does not appear to be directly related to blood and urinary steroid hormone levels. There is a low incidence of prostatic cancer among Mongoloids, with a relatively high incidence among Caucasians and some Negroes; the racial incidence shows considerable geographic variation. Prostatic cancer is rare before the age of 50 years, after which its incidence increases rapidly until the age of 80 years; thereafter, the incidence seems to drop. With regard to localizing factors, there is some indication that a type of atrophy associated with focal fibrosis may be followed by a precancerous hyperplasia. Benign enlargement of the prostate arises from the inner glands, while prostatic cancer arises within the outer glands. Invasion of the capsule occurs commonly and early and the tumor cells soon involve perineural lymphatics and blood vessels in the periprostatic tissues. There are two types of prostatic cancer which are morphologically indistinguishable: one behaves in the same way as any other clinical cancer, while the other remains inactive or latent. There are few reliable prognostic features, although penetration of the capsule and the presence of tumor cells in marrow biopsies are indisputable features associated with a poor prognosis. (30 references)

- 4215 RADIATION HAZARD CAUSED BY TECHNOLOGY.
(Ger.) Anonymous. *Umschau* 73(16):496-498, 1973.

Recent studies have demonstrated that genetic damage caused by radiation has been overestimated, while the hazard of radiation-induced cancer has been underestimated. Exposure to radiation from natural sources is estimated to cause less than 1-4 deaths/yr/million population, accounting for only 0.05-0.2% of the incidence of cancer (the cancer mortality in the Federal Republic of Germany is about 2350/yr/million population). The maximum permissible dose of artificial radiation (170 millirem/yr/person, which does not currently include radiation used in medical diagnosis and treatment) would add only 2-7 cancer deaths/yr/million population or increase the cancer risk by less than 1/1000 to 3/1000. If natural variation in the incidence of cancer is taken into account, a mean

dose of radiation from artificial sources, including medical radiation, of 170 millirem/yr/person would not appreciably increase the risk of cancer in the total population, providing that it does not exceed 100 million people. (No references)

- 4216 THE VIRAL LEUKEMIA-SARCOMA COMPLEX IN CATS. (Fr.) Wyers, M. (Natl. Veterinary Sch., Alfort, France). *Bull Mem Soc Med Paris* 176(6):46-49, 1973.

The morphology, physicochemical properties, *in vitro* replication, and carcinogenicity *in vivo* are considered for feline leukemia and feline sarcoma viruses. Although feline leukemia virus is secreted in the saliva, it has not been shown to be transmitted in this way. It is considered much more likely that this virus or its genome is transmitted from the mother to the fetus. Except for the dog, feline leukemia virus does not appear to be transmitted to man or other animals. (43 references)

- 4217 OCCUPATIONAL CANCERS. CURRENT AND FUTURE PROBLEMS. (Fr.) Dinman, B. D. (Inst. Environmental Industrial Hlth, U. Michigan, Ann Arbor). *Z Praeventivmed* 17(6):331-340, 1972.

This review briefly considers the use of animal experiments and epidemiological data to elucidate the roles played by various agents in occupational cancers. Agents considered to be definitely carcinogenic are β -naphthylamine, 4-aminobiphenyl, asbestos, mineral oil, tars, benzidine, and chromates. Our knowledge about wood dust, beryllium, 4-nitrobiphenyl, arsenic, α -naphthylamine, and hematite is far from adequate. Either there are no pertinent epidemiological data or animal experiments have given equivocal results. Substances which are carcinogenic in animals but for which there are no epidemiological data include lead, 4,4'-methylene-bis-o-chloroaniline, and 4,4'-diaminodiphenylmethane. (22 references)

- 4218 METASTATIC TUMORS OF THE ORAL SOFT TISSUES. REVIEW OF THE LITERATURE AND REPORT OF A CASE. (E.) Hatziotis, J. C. (Dept. Stomatology, Thessaloniki U., Greece), H. Constantinidou and P. H. Papanayotou. *Oral Surg* 36(4):544-550, 1973. (48 references)

- 4219 ON THE NATURE AND STRUCTURAL CHARACTERISTICS OF CANCER. GENERAL OBSERVATIONS ON THE MINUTE STRUCTURE OF MORBID GROWTHS. (E.) Müller, J. (No affiliation). *CA* 23(5):307-312, 1973. (No references)

- 4220 CRITERIA FOR HORMONE-DEPENDENT BREAST CANCER. (Rus.) Gnatyshak, A. I. (L'vov Med. Inst., USSR) and E. A. Gnatyshak. *Vopr Onkol* 19(7):97-108, 1973. (108 references)

- 4221 CYTOLOGICAL DIAGNOSIS OF ESOPHAGEAL CANCER. (Rus.) Galaktionov, V. V. (Leningrad Pediatric Med. Inst., USSR). *Vopr Onkol* 19(9):104-111, 1973. (54 references)

- 4222 CARCINOGENS OCCURRING IN THE ENVIRONMENT. (Hun.) Kertai, P. (Natl. Public Hlth. Inst., Budapest, Hungary). *Egeszsegstudomány* 17:297-309, 1973. (144 references)

- 4223 PRELEUKEMIAS. (Ger.) Heimpel, H. (Ctr. Internal Med. Pediatrics, U. Ulm, Germany) and J. Bauke. *Med Klin* 67(31):997-1003, 1972. (42 references)

- 4224 BURKITT'S LYMPHOMA. (E.) Burkitt, D. (Medical Res. Council, London, England). *Cancer Bull* 25(4):64-67, 1973. (4 references)

- 4225 ACUTE LYMPHATIC LEUKEMIA. (E.) Holland, J. F. (Roswell Park Memorial Inst., Buffalo, N.Y.). *Cancer Bull* 25(4):72-76, 1973. (3 references)

- 4226 HODGKIN'S DISEASE. (E.) DeVita, V. J., Jr. (Natl. Cancer Inst., Bethesda, Md.). *Cancer Bull* 25(4):77-79, 1973. (1 reference)

- 4227 HEMOSTASIS IN CANCER PATIENTS. (Rus.) Rakhmayeva, V. A. (Sci. Res. Inst. Clin. Exp. Surg. USSR Min. Publ. Hlth.), E. N. Vantsiyan, Ch. S. Guseynov and L. A. Petrov. *Vopr Onkol* 18(7):90-97, 1972. (60 references)

- 4228 VIRUSES AND LEUKAEMIA. (E.) Anonymous. *Br J Haematol* 25(3):287-291, 1973. (15 references)

- 4229 REPORT OF A MEETING. 7TH MEETING OF THE EUROPEAN TUMOR VIRUS GROUP ZIERIKZEE, NETHERLANDS, SEPTEMBER 25-27, 1972. (Ger.) Micheel, B. (Ctr. Inst. Cancer Res., Berlin-Buch, W. Germany) and E. Bender. *Arch Geschwulstforsch* 41(3):276-278, 1973. (No references)

- 4230 PRIMARY NEOPLASMS OF THE CENTRAL NERVOUS SYSTEM IN CHILDREN. (E.) Freeman, A. I. (Roswell Park Mem. Inst., Buffalo, N.Y.), N. K. Shah and R. Bourke. *Paediatrician* 1(4-5):239-248, 1972/1973. (30 references)

- 4231 CLASSIFICATION AND PATHOGENESIS OF HYPERPLASTIC AND PROLIFERATIVE DISEASES OF THE MESENCHYMA. (Fr.) Cagnoni, M. (Inst. Med. Symptomatology, U. Florence, Italy), L. L. Pozzi and B. Tarquini. *Lille Med* 18(2):173-178, 1973. (30 references)

- 4232 SQUAMOUS CELL "PAPILLOMAS" OF THE ORAL CAVITY, SINO-NASAL TRACT, AND LARYNX. (E.) Batsakis, J. G. (Dept. Path., U. Michigan, Ann Arbor) and H. A. Homburger. *Univ Mich Med Cent J* 38(3):111-119, 1972. (29 references)
- 4233 MULTIPLE MYELOMAS. I. GENERAL INFORMATION. (Fr.) Hoerni, M. B. (Bergonie Fdn., Bordeaux, France) and G. Hoerni-Simon. *Bordeaux Med* 5(11):1249-1254, 1972. (27 references)
- 4234 THE EPIDEMIOLOGY OF SOME CANCEROUS OR PARACANCEROUS DISEASES WHICH APPEAR TO BE OF INFECTIOUS ORIGIN. (Fr.) Boyer, J. (Inst. Hyg. Preventive Med., Paris, France). *Bull Acad Natl Med* 157(1):53-57, 1973. (No references)
- 4235 ODONTOGENOUS TUMORS. (Fr.) Brocheriou, C. (Pitie-Salpetriere Hosp., Paris, France), M. Auriol and G. Chomette. *Arch Anat Pathol* 20(2):203-222, 1972. (120 references)
- 4236 THE SUPRASELLAR MENINGIOMA. A REVIEW OF THE LITERATURE AND PRESENTATION OF A SERIES OF 31 CASES. (E.) Ehlers, N. (Aarhus U. Hosp., Denmark) and R. Malmros. *Acta Ophthalmol [Suppl] (Kbh)* 121:1-74, 1973. (172 references)
- 4237 "LEUKOPLAKIA," "KERATOSIS," AND INTRA-EPITHELIAL SQUAMOUS CELL CARCINOMA OF THE HEAD AND NECK. (E.) Batsakis, J. G. (Dept. Path., U. Michigan, Ann Arbor) and H. A. Homburger. *Univ Mich Med Cent J* 38(4):157-168, 1972. (5 references)
- 4238 CARCINOMA OF THE STOMACH. (E.) Lawrence, W., Jr. (Med. Coll. Virginia, Richmond). *CA* 23(5):286-304, 1973. (25 references)
- 4239 CELL CONTROL MECHANISMS. (E.) Anonymous. *Nature* 246(149):1, 1973. (No references)
- 4240 COMPARATIVE STUDY OF MUTAGENIC AND CARCINOGENIC ACTION OF CERTAIN CHEMICALS. (E.) Pogossianz, H. E. (Inst. Exp. Clin. Oncology, Acad. Med. Sci., Moscow, USSR). *Neoplasma* 20(5):527-530, 1973. (33 references)
- 4241 COMMON ORIGIN FOR ALL NEUROENDOCRINE TUMORS. (E.) Gammill, S. L. (Tennessee U. Sch. Med., Memphis) and R. Weichert. *Acta Radiol [Ther] (Stockh)* 12(4):321-326, 1973. (30 references)
- 4242 REGULATION OF ALPHA - FETOPROTEIN SYNTHESIS. (E.) Abelev, G. I. (N.F. Gamaleya Inst. Epidemiol. Microbiol., Acad. Med. Sci., Moscow, USSR). *Neoplasma* 20(5):563-566, 1973. (13 references)
- 4243 THE MECHANISMS OF SPREAD OF CANCER. (E.) Cole, W. H. (Asheville, N.C.). *Surg Gynecol Obstet* 137(5):853-871, 1973. (125 references)
- 4244 EXOGENOUS HORMONES - BOON OR CULPRIT? (E.) Wynder, E. L. (American Hlth Fdn., New York, N.Y.). *J Natl Cancer Inst* 51(3):729-731, 1973. (14 references)
- 4245 VIRILIZATION IN PREGNANCY COEXISTING WITH AN (OVARIAN) MUCINOUS CYSTADENOMA: A CASE REPORT AND REVIEW OF VIRILIZING OVARIAN TUMORS IN PREGNANCY. (E.) Verhoeven, A. T. M. (Dept. Obstetrics, Gynecology, U. Nijmegen, Netherlands), J. L. Mastboom, H. A. I. M. Van Leusden and W. H. M. Van Der Velden. *Obstet Gynecol Surv* 28(9):597-622, 1973. (104 references)
- 4246 OCCUPATIONAL ASBESTOSIS. (Fr.) Desbordes, J. (Calmette Hosp., Le Havre, France), J. Tayot, H. Duwoos, J. L. Ernoult, F. Manouvrier, L. Rousselin, P. Thoreux, J. Veret, R. Pingard and R. Bezannier. *Rev Tuberc Pneumol (Paris)* 36(8):1203-1222, 1972. (No references)
- 4247 MALE BREAST CANCER. I. HISTOLOGIC TYPING AND GRADING OF 187 DANISH CASES. (E.) Visfeldt, J. (Frederiksberg Hosp., Copenhagen, Denmark) and O. Scheike. *Cancer* 32(4):985-990, 1973. (22 references)
- 4248 NINE OUT OF TEN "DES BABIES" HAVE VAGINAL ADENOSIS. (E.) Anonymous. *Med World News* 14(41):17-19, 1973. (No references)
- 4249 PROLIFERATION AND DIFFERENTIATION OF GASTRO-INTESTINAL CELLS. (E.) Lipkin, M. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.). *Physiol Rev* 53(4):891-915, 1973. (197 references)
- 4250 NEUROBLASTOMA AND MYOCLONIC ENCEPHALOPATHY: TWO CASES AND A REVIEW OF THE LITERATURE. (E.) Senelick, R. C. (U. Utah Coll. Med., Salt Lake City), P. F. Bray, M. E. Lahey, H. J. L. VanDyk and D. G. Johnson. *J Pediatr Surg* 8(5):623-632, 1973. (29 references)
- 4251 IMMUNOLOGIC ASPECTS OF THE HEMATOLOGIC NEOPLASMS. (E.) Jones, S. E. (U. Arizona Coll. Med., Tucson), B. G. M. Durie and S. E. Salmon. *Postgrad Med* 54(5):209-216, 1973. (40 references)
- 4252 THE VIRAL FACTOR IN THE GENESIS OF BREAST CANCER: PRESENT EVIDENCE. (E.) Dmochowski, L. (Texas Med. Ctr., Houston). *Triangle* 12(2):37-47, 1973. (68 references)

4253 RECENT DEVELOPMENTS IN INDUSTRIAL CARCINOGENS. (E.) Eckardt, R. E. (Med. Res. Div., Esso Res., Engineering Company, Linden, N.J.). *J Occup Med* 15(11):904-907, 1973. (49 references)

4254 IMMUNITY STUDIES ON CANCER PATIENTS.
I. NONTUMORAL IMMUNITY. (Fr.) Cappelaere, P. (Oscar Lambret Ctr., France), L. Adenis and A. Demaille. *Lille Med* 18(1):96-112, 1973. (108 references)

4255 CANCER AND FOOD. (E.) Anonymous. *Lancet* (7838):1133-1134, 1973. (9 references)

4256 CADMIUM AND THE LUNG. (E.) Anonymous. *Lancet* (7838):1134-1135, 1973. (25 references)

4257 IMMUNITY STUDIES ON CANCER PATIENTS.
II. TUMORAL IMMUNITY. (Fr.) Cappelaere, P. (Oscar Lambret Ctr., France). A. Demaille and L. Adenis. *Lille Med* 18(2):179-201, 1973. (93 references)

- 4258 CARCINOGENICITY OF NITROSAMINES AND METHANESULPHONATE ESTERS GIVEN INTRAPERITONEALLY, IN RF MICE. (E.) Clapp, N. K. (Oak Ridge Natl. Lab., Tenn.). *Int J Cancer* 12(3):728-733, 1973.

The incidence of tumor formation in various organs of noninbred male RF mice was determined following single i.p. injections of various doses of the carcinogens dimethylnitrosamine (DMN), diethylnitrosamine (DEN), methyl methanesulphonate (MMS), or ethyl methanesulfonate (EMS). Animals were examined periodically throughout their lifetime and all major organs were examined grossly and histologically at autopsy. Based on mortality rates, DMN was the most carcinogenic substance. At 15 mg/kg, 75% of DMN-treated mice were dead at 30 days. DEN showed lower carcinogenic activity. MMS and EMS were not carcinogenic under the conditions of this study. DEN induced lung adenomas, liver hepatomas, forestomach squamous cell carcinomas, and papillomas at all concentrations studied, and induced Leydig-cell tumors of the testis at the highest dose (175 mg/kg). DMN increased lung tumor incidence but failed to induce liver tumors. None of the substances used in this study was leukemogenic. Comparison of the types of tumor induced following a single i.p. injection with those induced by repeated p.o. administration suggested that the route of administration had no effect on DEN carcinogenic activity. The absence of liver tumors following i.p. DMN injection, however, suggested an altered carcinogenic pattern for this substance.

- 4259 IS CANCER CAUSED BY BENZPYRENE IN NATURAL FERTILIZERS? (Ger.) Borneff, J. (Hyg. Inst. U. Mainz, Germany), H. Kunte, G. Farkasdi and H. Glathe. *Umschau* 73(20):626-268, 1973.

In field and pot trials it was demonstrated that the use of organic fertilizers (fresh and commercial compost, manure) did not increase the content of polycyclic aromatic hydrocarbons (PAH) in crops enough to cause an appreciable increase in the risk of cancer. Only underground parts of the plants were affected. The only way that all of the results could be explained is to assume that some of the PAH are synthesized and broken down by soil microorganisms, particularly bacteria and actinomycetes. Since the PAH content of plants increased to different extents, some but not all of the PAH found in plants could come from PAH present in the air as a pollutant. It is concluded that compost made from town trash and garbage may safely be used as fertilizer.

- 4260 THE CARCINOGENIC ACTION OF N-NITROSO COMPOUNDS. 1. N-NITROSO-3,6-DIHYDROOXAZINE-1,2 AND N-NITROSOTETRAHYDROOXAZINE-1,2. (Ger.) Wiessler, M. (German Cancer Res. Ctr., Heidelberg) and D. Schmahl. *Z Krebsforsch* 79(2):114-117, 1973.

Male and female Wistar rats were given 40-50 mg/kg/day of N-nitroso-3,6-dihydrooxazine-1,2 (NDO) or N-nitrosotetrahydrooxazine-1,2 (NTO) in their drinking water 6 days a week until the animals died or a total of 21.8 g/kg had been administered. Malignant

tumors developed in 5 of 20 rats given NDO and in 9 of the 20 given NTO. Tumors in NDO-treated rats consisted of subcutaneous spindle cell sarcomas (2), diffuse mediastinal carcinosis (1), hepatoma (1), and a mixed tumor of the left testicle (1). The average induction time was 660 ± 55 days. One female developed a benign mammary fibroadenoma. Malignancies found in NTO-treated rats consisted of: mammary adenocarcinoma associated with a pheochromocytoma of the left adrenal and pulmonary adenomatosis; an adenocarcinoma of the right orbit with multiple lung adenomas; myeloid leukemia; a squamous cell carcinoma of the nasal cavity which metastasized to the lung; a pulmonary carcinoid; blast cell leukemia; and three squamous cell carcinomas of the lung. The mean induction time for NTO was 440 ± 25 days. Benign tumors found in these rats consisted of multiple pulmonary adenomas, a papilloma of the paranasal sinuses, and a mammary fibroma. The lower carcinogenic activity of NDO is explained by hydroxylation of its double bond *in vivo* to form water-soluble nitrosamines which are excreted before the doses needed to induce cancer can build up in the body. The greater carcinogenicity of NTO cannot be accounted for simply by metabolic activation to the diazohydroxide and then to the methyl cation.

- 4261 THE CARCINOGENIC ACTION OF N-NITROSO COMPOUNDS. 2. S(+) AND R(-)-N-NITROSO-2-METHYLPIPERIDINE. (Ger.) Wiessler, M. (German Cancer Res. Ctr., Heidelberg) and D. Schmahl. *Z Krebsforsch* 79(2):118-122, 1973.

Optically pure S(+) and R(-) N-nitrosopiperidine were administered to male and female Sprague-Dawley rats by adding 30 mg/kg/day to their drinking water 6 days a week. After 8 months malignant tumors had developed in 15 of the 20 rats given the S(+) compound and in 12 of the 20 given the R(-) compound. The organotropism of their action, tumor induction times, and total doses needed for tumor induction were about the same in these two groups. The most common tumor in both groups of rats was ependymoblastoma of the olfactory nerve or bulb. Hepatocellular carcinomas developed in 3 rats given the S(+) compound but in none of those receiving the R(-) compound. However, the R(-) compound did induce three benign hepatocellular adenomas. Only one control developed a malignant tumor, a pheochromocytoma.

- 4262 RETENTION OF PARTICLES IN BRONCHIAL CELLS: POSSIBLE RELATIONS WITH INHALED CARCINOGENS. (Fr.) Masse, R. (Lab. Exp. Toxicol., Montrouge, France), P. Fritsch, R. Ducouso, J. Lafuma and J. Chretien. *C R Acad Sci (Paris) [D]* 276(21):2923-2925, 1973.

Tantalum powder was administered through a laryngeal catheter to 5 Wistar rats, 2 cats and 3 macaque monkeys, and hematite (iron oxide) powder (50 mg/g of the lung) was similarly administered to a baboon. Examination of the lungs 10 days later showed that tantalum powder was present in the small bronchi of monkeys. Much smaller amounts were found in rat bronchi, and no tantalum was present in cat bronchi.

A small number of tantalum particles were detected within cells of the bronchial epithelium of the cat, and some particles were found, particularly in cells of the primary ciliated bronchi, in the rat. Large numbers of tantalum particles were found in cells of the bronchial epithelium of the monkeys; they were even present in large cartilaginous bronchi. Electron microscope examination of sections of the baboon's lung revealed that hematite particles were present in the cytoplasm of bronchial cells, and small particles were found in some post-lysosomes. These particles occurred both in young mucus cells and in basal cells. Since no phagocytic vacuoles were observed, it is unlikely that tantalum or hematite enter bronchial cells by phagocytosis. Some evidence of pinocytosis was observed, but particle penetration might be purely passive. Since the number of intracellular particles was directly related to bronchial retention of these powders, this might account for the susceptibility of some species, particularly man, to bronchial carcinoma. This demonstrates the potential role of these particles as vectors of adsorbed carcinogens.

- 4263 DEVELOPMENT OF BLADDER CARCINOMAS IN RATS AND DOGS FED 1,2-DIHYDRO-2-(5'-NITROFURYL)-4-HYDROXYQUINAZOLINE-3-OXIDE. (Ger.) Engelbart, K. (Pharm. Sci. Lab., Farbwerke Hoechst, Frankfurt/Main, Germany), R. Brunk and E. Schutz. *Z Krebsforsch* 79(3):156-175, 1973.

1,2-Dihydro-2-(5'-nitrofuryl)-4-hydroxyquinazoline-3-oxide (DNHO), which effectively controls urinary tract infections, was administered p.o. to Wistar rats and beagles, and its effect on the bladder mucosa was investigated. Rats received 160, 1000 or 6300 mg/kg/day in a 2% starch suspension p.o. through a stomach tube for 12 or 26 weeks; in addition, 1600, 4000 or 10,000 ppm of DNHO were added to their food. In a short-term experiment rats received 10,000 ppm of DNHO for 2 or 3 weeks. Beagles received 50 or 100 mg/kg/day for 4 or 26 weeks. In both rats and dogs the earliest change observed was thickening of the epithelium in the bladder which was followed by localized or generalized squamous metaplasia. Microcarcinomas developed in 4 of 97 rats given the two highest doses of DNHO and in 2 of 11 beagles treated for 26 weeks. A few rats and dogs had papillomatous proliferation, and some rats developed hydronephrosis as a result of the bladder changes. It is recommended that all 5-nitrofur compounds be investigated experimentally for possible carcinogenic action on the mucosa of the bladder.

- 4264 CARCINOGENESIS BIOASSAY OF CHLORINATED DIBENZODIOXINS AND RELATED CHEMICALS. (E.) King, M. E. (Life Sci. Res. Div., IIT Res. Inst., Chicago, Ill.), A. M. Shefner and R. R. Bates. *Environ Health Perspect* 5:163-170, 1973.

Chlorinated dibenzodioxins were applied thrice weekly to the backs of Swiss-Webster and B6C3F1 mice and Osborne-Mendel rats, and were given orally in the daily food or water to Osborne-

Mendel rats and B6C3F1 mice. For the study of promotion activity, a group of mice in the skin application study was treated with dimethylbenzanthracene (DMBA) 1 week prior to the initiation of test compound application. In the skin application series: octachlorodibenzodioxin, dichlorodibenzodioxin, and acetone produced no significant pathology; and unsubstituted and dibenzodioxin and dioxane produced one malignant lymphoma each. In the promotion series: neither octachlorodibenzodioxin nor unsubstituted dibenzodioxin produced any significant pathology; croton oil produced neoplastic lesions of the skin and lungs; dichlorodibenzodioxin produced one plasmacytoma; acetone produced one papilloma and one malignant lymphoma; and dioxane produced neoplastic lesions of the skin, lungs, and kidney. In the oral administration studies, hepatotoxicity was the major effect noted, although one mouse exhibited broncheolar mucosal hyperplasia. Growth depression and mortality were widespread in the animals fed octachlorodioxin, with the male mice and female rats showing the greatest sensitivity.

- 4265 AUGMENTED INCIDENCE OF NEOPLASIA IN NZB/NZW MICE TREATED WITH LONG-TERM CYCLOPHOSPHAMIDE. (E.) Walker, S. E. (U. Michigan Med. Sch., Ann Arbor) and G. G. Bole, Jr. *J Lab Clin Med* 82(4):619-633, 1973.

Chronic administration of immunosuppressive drugs has been associated with an increased occurrence of neoplastic disease in certain clinical situations. In this study, the incidence of tumors was determined in cyclophosphamide-treated NZB/NZW mice (animal models of systemic lupus erythematosus). Neoplastic disease appeared in 94% of mice receiving "high dose" cyclophosphamide (8 mg/kg per day) compared to a tumor incidence of 21% in "low dose" treated mice (1 mg/kg per day) and 9% in control mice. In high-dose mice neoplasms developed after long periods of uninterrupted therapy, appearing at earlier ages in females compared to males. Generalized malignancies grew rapidly, causing death in 24 hr. An increased prevalence of benign tumors was found in older high-dose treated mice. In seven cases, multiple tumors appeared. Life-spans of high-dose female mice were prolonged significantly over life-spans of control female mice, but high-dose male mice had essentially the same longevity as male control mice. Low-dose cyclophosphamide had no effect on longevity or occurrence of tumors.

- 4266 CORRELATION BETWEEN SERUM PROLACTIN LEVELS AND INCIDENCE OF MAMMARY TUMORS INDUCED BY 7,12-DIMETHYLBENZ[a]ANTHRACENE IN THE RAT. (E.) Gala, R. R. (Wayne State U. Sch. Med., Detroit, Mich.) and S. J. Loginsky. *J Natl Cancer Inst* 51(2):593-597, 1973.

The administration of 7,12-dimethylbenz[a]anthracene (DMBA) (5 mg through the tail vein) to otherwise untreated control female Sprague-Dawley rats, 52 days old, induced mammary tumors with an average latency period of 79.2 ± 2.2 days in all animals. Only 60% of

the rats given DMBA, followed by the taking of daily vaginal smears and weekly blood sampling, under heavy ether anesthesia developed tumors. The average latency time of this group was 93.4 ± 4.5 days. The difference between the groups in latency time was statistically significant ($P < 0.05$). Tumor regression for experimental animals was 40% and 18% for control animals. Ether-stressed serum prolactin levels were significantly higher ($P < 0.001$) in animals not developing tumors than in those that did. At each stage of the estrous cycle the serum prolactin level was higher for animals without tumors. The data indicate that animals with the capacity to secrete high levels of prolactin, under ether stress, are resistant to DMBA-induced mammary tumors. Whether this is a mechanism related specifically to prolactin or involves other hormones is not known, but prolactin appears to be involved in protecting the animals against tumor development.

- 4267 MORPHOLOGICAL AND AUTORADIOGRAPHICAL INVESTIGATIONS ON EXPERIMENTAL CARCINOGENESIS AND POLYP DEVELOPMENT IN THE INTESTINAL TRACT OF RATS AND MICE. (E.) Wiebecke, B. (Inst. Path., U. Munich, Germany), U. Krey, U. Lohrs and M. Eder. *Virchows Arch (Pathol Anat)* 360:179-193, 1973.

Most types of neoplastic lesions seen in human intestinal mucosa were reproduced experimentally in rats and mice after application of 1,2-dimethylhydrazine (DMH). The rats (50 female Wistar) received DMH s.c. in doses of 14 or 21 mg/kg body wt each week; the mice (40 female NMRI) received a weekly s.c. dose of 14 mg/kg body wt. Animals were killed beginning with the third month of the experiment. Tumor distribution revealed species specific and dosage dependent differences. In rats the tumors of the large bowel were mostly isolated and often short-stalked, whereas mice tended to develop broad-based and widespread tumors. In mice treated with 14 mg DMH/kg the descending colon inclusive the rectum was much more often affected than the ascending colon, whereas in similarly treated rats the frequency of tumor development was nearly the same in these two bowel segments. In animals treated with 14 mg DMH/kg, tumors of the small bowel were rare compared with the colon, but their number rose considerably after treatment with 21 mg DMH/kg. Primary nonpolypous carcinomas with early infiltration developed in the rat as a consequence of rapid epithelial dedifferentiation and malignant transformation, predominantly in the small intestine. Benign and malignant polypous tumors were much more frequent in the large than in the small intestine. Benign adenomatous and villous polyps of the colon develop after preceding mucosal hyperplasia due to partial loss of epithelial differentiation. As demonstrated by quantitative autoradiographical findings, the proliferation zone in these lesions is extended to the mucosal surface. Additionally, the zone of maximal epithelial proliferation is transposed to a mucosal layer near the surface. This qualitative change in the mode of epithelial proliferation compared with normal and hyperplastic mucosa is the essential factor in the development of benign neoplastic polyps.

- 4268 ENHANCEMENT OF SPONTANEOUS HEPATIC TUMORIGENESIS IN C3H MICE BY DIETARY PHENOBARBITAL. (E.) Peraino, C. (Argonne Natl. Lab., Illinois), R. J. M. Frey and E. Staffeldt. *J Natl Cancer Inst* 51(4):1349-1350, 1973.

Male and female C3H mice at 1 or 3 months of age, housed 1/cage or 5/cage, were fed a control diet or one containing 0.05% phenobarbital. After 12 months the mice were examined for spontaneous liver tumors. In mice fed the control diet, tumor incidence was higher in males than in females, and higher in mice housed 1/cage than in those housed 5/cage. Phenobarbital in the diet increased the incidence of liver tumors, irrespective of population density or sex, but did not decrease the degree of differentiation of the tumors. It is believed that phenobarbital only accelerates the expression of a previously induced tumorigenic lesion without increasing the severity of the lesion.

- 4269 TOXICITY OF CIGARETTE SMOKE COMPONENTS: FREE LUNG CELL RESPONSE IN ACUTE EXPOSURES. (E.) Rylander, R. (Natl. Environment Protection Board, Karolinska Inst., Stockholm, Sweden). *Am Rev Respir Dis* 108(5):1279-1282, 1973.

The effect of short-term exposure to cigarette smoke on the number of free lung cells was studied in guinea pigs. The animals received smoke from five cigarettes in small intermittent doses, with a resting period between each puff, in volumes and intervals that were as closely related to human smoking as possible. The number of macrophages and leukocytes was determined in lung lavage preparations two hr afterwards. A decrease in the number of free lung cells was found. The decreases were related to the composition of various types of tobacco tested, revealing a high negative correlation between the macrophage count and acetaldehyde and tar content. Acetaldehyde accounted for 63% of the effect on macrophages. The leukocyte response was most highly correlated with acetaldehyde and the pH of the particulate phase, acetaldehyde and pH accounting for 75% and 8.7%, resp., of the decrease. The free lung cell response probably represents a significant exposure reaction in terms of development of pulmonary disease.

- 4270 CARCINOGENICITY OF STERIGMATOCYSTIN TO RAT SKIN. (E.) Purchase, I. F. H. (S. African Med. Res. Council, Pretoria) and J. J. Van der Watt. *Toxicol Appl Pharmacol* 26(2):274-281, 1973.

Sterigmatocystin, a mycotoxin which is carcinogenic when given orally to rats, was tested for its carcinogenic effects on rat skin. The shaved skin of 40 male rats from a Wistar-derived strain was treated with 1 mg of sterigmatocystin in dimethyl sulfoxide or in acetone twice weekly for 70 wk. A group of 10 rats served as the untreated control. At 40 wk papillomas developed, and by 70 wk all treated rats had either papillomas or squamous cell carcinomas. In 17 out of the 20 rats treated with sterigmatocystin, liver lesions, consisting of regenerative changes or hepatocellular carcinomas, were present.

The observation that sterigmatocystin is carcinogenic to rat skin emphasizes the danger of skin contamination to laboratory workers.

- 4271 CELLULAR IMMUNITY IN MICE CHRONICALLY EXPOSED TO FRESH CIGARETTE SMOKE. (E.) Thomas, W. R. (Perth Med. Ctr., Western Australia), P. G. Holt and D. Keast. *Cell Immunol* 27(6): 372-375, 1973.

Inbred C57 Black mice were exposed daily to fresh cigarette smoke for 5 to 35 weeks. Cell-mediated immunity, measured by the phytohemagglutinin (PHA) responsiveness of lymphocytes from various organs, was determined at different times during exposure. An initial enhancement was noted in the PHA responsiveness of lymphocytes from regional lymph nodes of the respiratory system, but by the 35th week of exposure the response was markedly depressed. Lymphocytes recovered from the blood and spleen reacted similarly, except that at the end of the 35th week only the peak response in the blood was significantly depressed. The similarity in responses of the blood and spleen reflects not only the circulatory nature of the PHA responsive cell, but also the systemic effect of prolonged exposure to cigarette smoke. It is suggested that the loss of PHA responsiveness of lymphocytes following exposure to cigarette smoke may increase the potential for tumorigenesis by a mechanism unrelated to the deposition of carcinogens in the respiratory tract.

- 4272 ACTION OF AIR POLLUTANTS AND POLYCYCLIC HYDROCARBONS ON THE RESPIRATION OF FETAL CALF-KIDNEY CELL CULTURE. (E.) Bottex, C. (Army Health Dept., Lyon, France), R. Giordani, P. Deschaux and R. Fontages. *J Natl Cancer Inst* 51(4):1129-1133, 1973.

The action of atmospheric air pollutants (or their extracts) and some polycyclic synthetic hydrocarbons on the metabolism of fetal calf-kidney cell cultures was investigated at several concentrations as a function of exposure time. At first, the tested agents inhibited respiratory exchanges. However, after a 72-hour treatment of cultured cells with an organic extract or some polycyclic synthetic hydrocarbons, some respiratory hyperactivity was observed. The two most active hydrocarbons were benzo(a)pyrene and benz(a)anthracene. Benzo(e)pyrene exhibits moderate activity. The question is raised whether the respiratory increase observed 72 hr after treatment might not mark the accession of cell cultures to a pretumorous phase.

- 4273 RAPID VIRAL INDUCTION OF PLASMACYTOMAS IN PRISTANE-PRIMED BALB/c MICE. (E.) Potter, J. (Natl. Inst. Hlth., Bethesda, Md.), M. D. Sklar and W. P. Rowe. *Science* 182(4112):592-594, 1973.

Strain BALB/c mice were injected i.p. with 0.5 ml of pristane and injected 39 to 56 days later with Rous sarcoma virus (MLV-A), a lymphosar-

comagenic variant of Moloney virus. Twenty-four (58%) of 92 mice developed lymphosarcoma, and 26 (28%) developed immunoglobulin-producing plasmacytomas within 20 to 93 days (77 to 149 days after the pristane injection). Two (3.5%) of 57 control mice developed plasmacytomas at days 138 and 166 after a single injection of pristane; no plasmacytomas were found in mice treated with virus alone. MLV-A infection may induce transformation of plasma cells or their precursors, while the role of pristane might be either to increase the number or susceptibility of the target population, or to induce microenvironmental changes essential for growth of detectable plasmacytomas. The marked reduction in the induction time for plasmacytomas using this system will facilitate determination of factors involved in the pathogenesis of plasmacytomas.

- 4274 DIETARY COPPER AND THE INDUCTION OF NEOPLASMS IN THE RAT BY ACETYLAMINOFLUORENE AND DIMETHYLNITROSAMINE. (E.) Carlton, W. W. (Sch. Vet. Sci. Med., Purdue U., Lafayette, Ind.) and P. S. Price. *Food Cosmet Toxicol* 11(5):827-840, 1973.

The effect of dietary copper on the incidence and location of neoplasms was studied in rats fed copper-deficient (1 ppm) and excess-copper (800 ppm) diets containing acetylaminofluorene (AAF) and in rats fed the same copper-deficient and excess-copper diets and given dimethylnitrosamine (DMN) in the drinking-water for 9 months. The excess copper with or without DMN or AAF was toxic, body-wt gains being reduced and mortality increased. Reduction in wt gain was greatest in the excess-copper-AAF group, but the highest mortality (72%) occurred in the excess-copper-DMN group. Liver wt was increased in the AAF-treated rats, partly because of the presence of neoplasms. The average copper content (4.5 ppm) in hepatic tissue from rats fed the copper-deficient diet was greater than that found in hepatic tissue from DMN-treated (3.9 ppm) or AAF-fed rats (2.8 ppm). Hepatic copper concentrations of rats fed the excess-copper diet averaged 244 ppm in controls, while the comparable figures for the DMN-treated and AAF-fed rats were 394 and 354 ppm respectively. Neoplastic hepatic tissue from rats fed the copper-deficient diet had a copper content similar to that of the non-neoplastic tissue, but the neoplastic hepatic tissue from rats fed the excess-copper diet was lower than that of non-neoplastic liver. Neoplastic tissue from the kidney had less copper than grossly normal renal tissue. The incidence of hepatic neoplasms was similar in DMN-treated rats fed either the copper-deficient or excess-copper diets, but kidney neoplasms occurred in 57% of the rats receiving the copper-deficient-DMN treatment and killed for autopsy, compared to an incidence of 0.0% in the excess-copper-DMN group. This difference in incidence may be related to the copper levels of the diet. The incidence of DMN-induced neoplasms in the lungs was similar in rats fed the copper-deficient and excess-copper diets. More AAF-fed than DMN-treated animals developed hepatic neoplasms, but dietary copper concentration did not have any effect on the incidence. Extra-hepatic neoplasms in AAF-fed rats occurred in the

lungs, spleen, skin, intestine, pancreas and muscle. The incidence of extrahepatic neoplasms was 40% in copper-deficient-AAF rats killed for autopsy, but only 17% in those fed the excess-copper diet.

4275 TUMORS INDUCED IN MICE WITH SMALL DOSES OF N-METHYLBENZYL-NITROSAMINE. (Ger.)

Sander, J. (Hyg. Inst., U. Tübingen, Germany) and F. Schweinsberg. *Z Krebsforsch* 79(3):157-161, 1973.

Experiments were performed on female NMRI mice to determine whether very small doses of methylbenzyl-nitrosamine (MBNA) could induce tumors. Of 100 mice given drinking water containing 20 ppm MBNA for 20-80 days (total dose of 11-44 mg/kg), all 100 developed tumors of the esophagus and forestomach and 5 developed pulmonary adenomas, as did 15 of the 60 controls. Invasive esophageal carcinomas developed in 16 of 60 mice given a total of 44 mg/kg MBNA, in 2 of the 20 given 22 mg/kg, and in 2 of 20 given 11 mg/kg; the other 80 mice had esophageal papillomas. Of 80 mice given drinking water containing 5 ppm MBNA for 10-80 days (total doses of 1.4-11 mg/kg), 51 had esophageal tumors, 28 tumors of the forestomach, and 28 pulmonary adenomas. More mice in this group had papillomas, rather than carcinomas, of the esophagus and forestomach than mice given drinking water containing 20 ppm MBNA. Carcinomas of the esophagus developed in 2 of 20 mice given a total of 11 mg/kg MBNA, but in 0 of 60 given a total of 1.4-5.5 mg/kg MBNA. Carcinomas of the forestomach were found in 12 mice given a total of 11 mg/kg MBNA, in 4 of 20 given a total of 5.5 mg/kg MBNA, in 5 of 20 given a total of 2.75 mg/kg MBNA, and in 0 of 20 given a total of 1.4 mg/kg MBNA. Of 140 mice given i.p. injections of MBNA in physiological saline (1-23 doses of 1.5-2.5 mg/kg; totals of 2.25-27.5 mg/kg), 92 had tumors of the forestomach (primarily papillomas) and 86 had pulmonary adenomas; none of these mice developed esophageal tumors. Since even the smallest doses of MBNA used in this study induced tumors, MBNA is a more powerful tumorigenic agent than was previously demonstrated. Since there was no clear delimitation of tumors from healthy tissue or from one another, it was impossible to compare the number of tumors induced with the dose of MBNA. With the exception of 2 groups, the latent time for tumor development increased and fewer animals developed tumors as doses of MBNA decreased. The exceptions to this rule can be attributed to the small number of mice in each group (20).

4276 ULTRASTRUCTURE OF EXPERIMENTAL BRAIN GLIOMAS IN MICE. (E.) Kroh, H. (Exp.

and Clin. Med. Res. Ctr., Polish Acad. Sci., Warsaw), T. Majdecki and K. Renkawek. *Z Krebsforsch* 80(2): 159-168, 1973.

The ultrastructural characteristics of 12 gliomas produced by the intracerebral implantation of methylcholanthrene were studied by electron microscopy. The fine structure of these gliomas did not differ significantly from that of gliomas cultured *in vitro*,

although the tumors produced by tissue culture had more nuclear membrane indentations than the corresponding cells produced *in vivo*. The nuclei contained large amounts of chromatin which flowed out through the distended pores of the nuclear membrane into the cytoplasm, and gliofibrils and microtubules were found in all types of gliomas. The experimental gliomas also contained highly developed and frequently modified Golgi apparatus, which occupied a large part of the cytoplasm in the malignant glioma and ependymoma. In addition, the endoplasmic reticulum of the malignant gliomas enlarged and became cystic, giving the cytoplasm of the tumor cells a spongy appearance. In the cytoplasm of some cells of astrocytomas and gliosarcomas from the *in vitro* material exclusively, particles with a characteristic structure resembling virus inclusions of RNA type were observed. Virus-like bodies were observed in the cytoplasm and nuclei of a few glioma cells: they were agglomerated in groups of several to several score.

4277 CARCINOGENIC, HEMATOTOXIC, AND HEPATOTOXIC EFFECTS OF ORGANOPHOSPHORUS PESTICIDES.

(Ger.) Gibel, W. (Central Inst. Cancer Res., Berlin-Buch, Germany), K. Lohs, G. P. Wildner, D. Ziebarth and R. Stieglitz. *Arch Geschwulstforsch* 41(4): 311-328, 1973.

Male and female Wistar rats, aged 10 wk, were given trichlorfon (15 mg/kg p.o. or i.m. twice a week) or dimethoate (5, 15, or 30 mg/kg p.o. or 15 mg/kg i.m. twice a week). Both pesticides were applied topically to the skin of male and female AB mice twice a week for 6 wk. A variety of malignant tumors developed in 11 of 55 rats given trichlorfon and in 15 of 101 given dimethoate; none of the controls developed malignant tumors. Benign tumors were diagnosed in 26 of the 55 rats given trichlorfon and in 19 of the 101 rats given dimethoate; 7 controls in each group had benign tumors, all of which were mammary fibroadenomas. The most common malignancies were reticulum cell sarcomas of the spleen (4 in rats given trichlorfon, 6 in rats given dimethoate) and malignant reticulosis (2 in rats given trichlorfon, 3 in those given dimethoate). Many of the rats had myeloproliferative changes which can be regarded as preleukemic. Benign tumors consisted of 23 papillomas or papillomatoses of the forestomach, 1 adenoma of the glandular stomach, 5 multiple adenomas of the lungs, 7 mammary fibroadenomas, 5 cholangiomas, 2 ovarian cysts, and 3 luteomas. Papillomatoses of the forestomach occurred only after p.o. administration of both pesticides. Myeloid leukemia developed in 5 of 14 mice given topical applications of trichlorfon. Dimethoate induced 5 malignancies in 19 mice; 4 were leukemia and 1 a mammary carcinoma.

4278 BEHAVIOR OF AFLATOXINS IN SOME MEAT PRODUCTS. (E.) Strzelecki, E. L. (Vet.

Hyg. Res. Stn., Gdansk, Poland). *Acta Microbiol Pol [B]* 5(22):171-177, 1973.

The growth of *Aspergillus flavus* and subsequent pro-

ductions of aflatoxins were studied in raw and country cured ham and salami. Meat samples injected or sprayed with a solution of *A. flavus* spores were tested after various storage periods and temperatures. Only trace amounts of toxin were detectable in cured ham after 126 days of storage. Raw ham contained no detectable toxins. Large amounts of toxin (105 and 213 µg/100 gm meat) were found in salami after 13 and 78 days of storage. The recovery of aflatoxins from meats inoculated with known amounts decreased with increasing time and was greatest for salami (19% after 78 days). Potassium sulfite, potassium fluoride, and p-aminobenzoic acid inhibited aflatoxin production in both ham and salami. Curing mixture (sodium chloride, potassium nitrate, sodium nitrite, and 2% yeast extract in water) produced a 2000-fold stimulation in the production of aflatoxins by *A. flavus* cultures when compared with control cultures grown in 2% yeast extract. Potassium nitrate alone was totally inhibitory, whereas sodium nitrite was slightly stimulatory. Twice as much aflatoxin G₁ as B₁ was recovered from curing medium-stimulated cultures. Certain other mold strains also isolated from cured ham (e.g. *A. ochraceus*) inhibited production of aflatoxins by *A. flavus*.

- 4279 TISSUE AND SUBCELLULAR DISTRIBUTION AND BINDING OF [¹⁴C]-METHYLCHOLANTHRENE OR ITS DERIVATIVES TO CELL CONSTITUENTS IN MATERNAL, FETAL AND NEONATAL MICE. (Fr.) Malaveille, C. (Intl. Ctr. Cancer Res., Lyon, France), B. Duperay, H. Pacheco and L. Tomatis. *Chem Biol Interact* 7(2):79-92, 1973.

The distribution of 3-methylcholanthrene (MCA) and/or its metabolites was investigated in various organs of maternal, fetal, and neonatal mice 6 and 60-66 hr after a single dose of 1 mg ¹⁴C-MCA had been administered p.o. through a stomach tube to pregnant CF-1 mice. The quantity of ¹⁴C-MCA and/or its metabolites bound to nuclear and cytoplasmic proteins was determined in organs which have been shown to be either susceptible (lung) or nonsusceptible (kidney) to the carcinogenic action of MCA. A slightly larger quantity of free and loosely bound MCA was found in subcellular fractions from lungs than in those from kidneys, and a larger quantity of labeled MCA was bound to nuclear DNA in the lungs than in the kidneys.

- 4280 BIOLOGICAL EFFECT AND HYGIENIC EVALUATION OF CYCLAMATES. (Hun.) Pinter, I. (Natl. Inst. Food Nutrition, Budapest, Hungary) and P. Czuczy. *Élelmiszervizsgálati Közlemények* 19(1-2):29-34, 1973.

Studies on the acute and chronic toxicity as well as on the carcinogenic effect of cyclamates, first licensed for use as sweeteners in the USA in 1950, are reviewed. Chromosome breaks due to cyclamates and their metabolite, cyclohexylamine, were found in rat and in human lymphocytes, skin fibroblasts, and laryngeal cancer cells. The first indication of a possible carcinogenic effect of cyclamates was found in 1969 when mice developed cancer of the

bladder in 16 months following placement of cholesterol balls containing 20% cyclamates in their bladders. Eight out of 80 rats having been fed 10:1 ratio of 2,500 mg of cyclamate/kg with saccharin developed cancer of the bladder. Cancer was not detected in a control group or in other rats fed 500 or 1,000 mg of cyclamate/kg. A slight, but distinct carcinogenic effect of cyclamates was detected in susceptible strains of mice that were fed 800 to 1,250 mg of cyclamate/kg during their lifetime starting from the 30th day of life. An epidemiological survey conducted in the USA in 1967 with the aim of establishing a possible relationship between the occurrence of cancer of the bladder in the population and cyclamate consumption was premature since the latency time of cancer in the human organism amounts to some 20 yr. In view of the rather scarce evidence of the carcinogenic effect of cyclamates, its use has been prohibited in the USA, and limited in Hungary.

- 4281 STUDIES ON THE MORPHOLOGY OF EXPERIMENTAL TUMORS INDUCED IN RATS BY INTRAPERITONEAL INJECTION OF ASBESTOS DUSTS. (Ger.) Scheuer, E. (Pathol. Inst., Univ. Dusseldorf, Germany), F. Huth and F. Pott. *Arch Geschwulstforsch* 41(2):120-136, 1973.

Since some forms of asbestos contain benzo(a)pyrene (BP) as an impurity, experiments were performed to determine whether BP had any effect on the carcinogenicity of asbestos or the type of tumors it induces. At one week intervals female Wistar rats received 4 i.p. injections of 25 mg asbestos dust (amosite, anthophyllite, crocydolite, chrysotile A, or finely ground chrysotile A) suspended in 2 ml physiological saline. Other rats received 1.25 ml of BP with or without asbestos. Rats were sacrificed and examined for tumors when they lost weight or developed jaundice. After 4-24 months, extensive, usually multicentric, abdominal tumors had developed in 215 of 420 rats. Five types of tumors were diagnosed: fibrosarcomas (107 cases), polymorphocellular sarcomas (68 cases), mesotheliomas (34 cases), reticulum cell sarcomas (2 cases), and adenocarcinomas (4 cases). Only 3 of 30 rats given BP alone had developed tumors (polymorphocellular sarcomas of the pancreas, greater omentum, and abdominal wall, resp.). Administration of asbestos increased the incidence of tumors about 50% in rats given BP. However, BP had no effect on the incidence of tumors or histological types of tumors induced by asbestos. The histological type of tumor induced by asbestos was independent of the form of asbestos administered.

- 4282 BLADDER CANCER AND *N*-METHYL-*N*-NITROSOUREA. II. SUB-CELLULAR CHANGES ASSOCIATED WITH A SINGLE NONCARCINOGENIC DOSE OF MNU. (E.) Wakefield, J. St. J. (Middlesex Hosp. Med. Sch. London, England) and R. M. Hicks. *Chem Biol Interact* 7(3):165-179, 1973.

The ultrastructural changes in the bladder epithelium of male Wistar rats induced by a single non-

carcinogenic but cytotoxic i.v. dose of *N*-methyl-*N*-nitrosourea (MNU, 2.0 mg), were studied and compared with some aspects of the bladder epithelial response to a single i.p. dose of cyclophosphamide (200 mg/kg). The initial sequence of events seen in the transitional epithelium were similar for both substances. Both produced an accumulation of cytoplasmic lipid cell edema, followed by necrosis, desquamation of the epithelium, and hemorrhage from congested sub-epithelial capillaries. Breaks in the basal lamina were commonly seen following cyclophosphamide treatment but were rare after MNU. After two wk, the bladder epithelium of MNU-treated rats became hyperplastic, increasing from a normal thickness of three cells to eight to ten cells. Bladders from cyclophosphamide-treated rats were not studied after two wk. All cell layers of MNU-treated bladder showed an increased number of cytoplasmic filaments which frequently aggregated into bundles to form tonofibrils. All changes began to reverse themselves from about three wk on, with reestablishment of normal, although slightly hyperplastic, epithelium in necrotic areas.

- 4283 IMMUNOSUPPRESSION AFTER SINGLE AND MULTIPLE PULSE DOSES OF 7,12-DIMETHYLBENZ[*a*]ANTHRACENE IN THE RAT. (E.) Sugiyama, T. (Kobe Univ. Sch. Med., Japan), T. O. Yoshida and Y. Nishizuka. *Gann* 64(4):397-400, 1973.

Male Long-Evans rats were given 7,12-dimethylbenz[*a*]anthracene (DMBA), i.v., as a liquid emulsion in either a single injection of 30 mg/kg or in a series of 4 injections of 30, 25, 25, and 25 mg/kg at 10 day intervals. Injections began at 28 days of age. After single DMBA injection the number of plaque forming cells per spleen decreased from about 10^5 to a minimum of $1-2 \times 10^3$ on day 15. Restoration then occurred reaching normal levels by day 60. Pulse injections of DMBA caused a sustained depression, at 1/20 that of the normal level. In 21 animals kept alive for a longer time, 8 developed leukemia between day 50 and 150. The number of plaque forming cells in these leukemic animals was extremely low, decreasing as leukemia developed. However, no direct correlation was determined between the number of plaque forming cells and the start of individual leukemia. The very low number of the cells in the leukemic spleen may be due to immunocompetent cell depletion due to leukemic infiltration. The results of this study indicate that multiple injections are needed to provide a better circumstance for the survival and proliferation of leukemic cells.

- 4284 DNA SYNTHESIS OF THE URINARY BLADDER EPITHELIUM IN RATS WITH LONG-TERM FEEDING OF *dl*-TRYPTOPHAN-ADDED AND PYRIDOXINE-DEFICIENT DIET. (E.) Miyakawa, M. (Kyoto U. Sch. Med., Japan) and O. Yoshida. *Gann* 64(4):411-413, 1973.

Male Wistar rats (16) were fed a pyridoxine deficient and *dl*-tryptophan supplemented diet for 56 wk. An additional seven rats served as controls. Tritiated thymidine (1 μ Ci/g, i.p.) was administered 1 hr before sacrifice. Microscopic examination showed

no bladder tumor or hyperplasia in the 16 experimental or 7 control rats. A total of 984 out of 83,710 bladder epithelial cells counted were labeled and the labeling index of this experimental group was 1.16%. The labeling index of the seven control rats was 0.06% and only 24 out of 39,799 cells were labeled. Histological examination showed the normal transitional epithelium of the bladder and no mitotic figures were noted for either group. It is suggested that the effect of tryptophan metabolites is related not only to the *l*-isomer, but also the *d*-isomer. It is also suggested that increased tritiated thymidine uptake may have been due not to tryptophan metabolites but due to the effect of pyridoxine deficiency on DNA synthesis.

- 4285 SUBCELLULAR LOCALIZATION OF HUMAN PLACENTAL ARYL HYDROCARBON HYDROXYLASE. (E.) Juchau, M. R. (U. Washington Sch. Med., Seattle) and E. A. Smuckler. *Toxicol Appl Pharmacol* 26(2):163-179, 1973.

Homogenates of human placentas were fractionated by a variety of differential centrifugation techniques. Prepared subfractions were assayed by ultrastructure analysis and marker enzyme quantitation. The lowest ratios of cytochrome oxidase/aryl hydrocarbon hydroxylase activities were observed in a 6.24×10^6 g-min sediment fraction following sequential precentrifugations at 1.5×10^4 , 9.75×10^4 , 2.18×10^5 , 3.03×10^5 and 4.64×10^5 g-min. Investigations of subfractions with respect to acid phosphatase, glucose-6-phosphatase, nucleoside diphosphatase, glucose-6-phosphate dehydrogenase, and aryl sulfatases A, B, and C also tended to support a concept of localization of the mixed-function oxidase system in the endoplasmic reticulum of human placental cells, and indicated that the membranes of the endoplasmic reticulum were the predominant site of 3,4 benzpyrene hydroxylation. Consistent with these chemical observations was the enrichment of membranous components seen in electron micrographs of the respective homogenate subfractions. Preliminary evidence also suggests that whereas placental aryl hydrocarbon hydroxylase activity is increased by cigarette smoking, aromatase and cholesterol side-chain oxidase activity are not increased; in fact, the latter appears to be decreased.

- 4286 INDUCTION OF LIVER TUMOURS IN RATS BY A SINGLE TREATMENT WITH NITROSO COMPOUNDS GIVEN AFTER PARTIAL HEPATECTOMY. (E.) Craddock, V. M. (Med. Res. Council Lab., Surrey, England). *Nature* 245(5425):386-388, 1973.

Rats underwent partial hepatectomy and were given an i.p. injection of dimethylnitrosamine (DMN, 4.8-16.8 mg/kg), diethylnitrosamine (DEN, 50-200 mg/kg), or methyl methanesulphonate (MMS, 22-110.5 mg/kg). An additional group of rats received *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine (MNNG, 10-25 mg/kg) by stomach tube. Drugs were administered at 0-2 hr, 3-5 hr, or 24 hr after hepatectomy. After operation, single doses of DMN produced liver carcinoma. The most effective time of dosing was

24 hr after operation, perhaps because of a variation in the cell sensitivity at different stages in the cell cycle; the 24 hr mark is the time of maximum DNA synthesis. DEN, similarly produced liver carcinomas after the operation but was effective at 2 hr perhaps because the DEN, taking longer to be metabolized, was still present at 24 hr. MNNG similarly induced liver cell cancer after operation. NMU has not thus far induced liver cancer, perhaps because most animals treated with this drug die of more rapidly growing tumors before 1.5 to 2 yr which is the time required for the development of liver tumors induced by the nitrosamines and MNNG. These results suggest that for carcinogenesis to result from treatment with alkylating agents, there must be damage of a special type to the genetic material and there must be cell replication before the damage has been repaired.

- 4287 ACTIVATION OF THE CARCINOGEN N-HYDROXY-2-FLUORENYLACETAMIDE: INSENSITIVITY TO CYANIDE AND SULFIDE OF THE PEROXIDASE-H₂O₂ INDUCED FORMATION OF NUCLEIC ACID ADDUCTS. (E.) King, C. M. (Michael Reese Hosp. Med. Ctr., Chicago, Ill.), T. W. Bednar and E. M. Linsmaier-Bednar. *Chem Biol Interact* 7(3):185-188, 1973.

The effect of specific enzyme inhibitors on the *in vitro* peroxidase-catalyzed formation of arylamine-nucleic acid conjugates was studied using ¹⁴C-labeled N-hydroxy-2-fluorenylacetamide (HO-FAA). Incubation of HO-FAA with purified horseradish peroxidase and yeast sRNA resulted in a significant amount of binding of ¹⁴C to nucleic acid as determined by liquid scintillation spectrometry. Addition of the peroxidase inhibitors cyanide or sulfide (10⁻² to 10⁻⁴ M) abolished peroxidase activity but had no significant effect on the activation and RNA binding of HO-FAA. Whether the prosthetic group of the peroxidase was functioning as an elaborate inorganic catalyst, or whether activation of HO-FAA was a result of a new enzymatic activity not previously recognized for the peroxidase molecule, could not be determined.

- 4288 DNA REPAIR SYNTHESIS AND SURVIVAL OF REPAIR DEFICIENT HUMAN CELLS EXPOSED TO THE K-REGION EPOXIDE OF BENZ(a)ANTHRACENE. (E.) Stich, H. F. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada) and R. H. C. San. *Proc Exp Biol Med* 142(1):155-158, 1973.

Cell cultures of fibroblasts were obtained from skin-punch biopsies taken from a patient with xeroderma pigmentosum (XP) and control persons of comparable age. The cultured cells were then exposed to the precarcinogen benz(a)anthracene (BA), its highly reactive K-region epoxide, and its metabolite cis-5,6-dihydrodiol. The active BA-epoxide induced DNA repair synthesis in the normal cells, but had only a slight effect on the level of DNA repair synthesis in the XP fibroblasts; no detectable DNA repair synthesis was triggered by BA or A-cis-5,6-dihydrodiol. The BA-epoxide also had a strongly lethal effect on the normal and XP

cells, while BA and BA-dihydrodiol did not. Compared with the normal cells, the XP cells exhibited an increased sensitivity to the BA-epoxide as measured by their clone-forming capacity. The response of the XP cells to the BA-epoxide resembles that following UV-irradiation or treatment with the oncogenic 4-nitroquinoline-1-oxides or N-acetoxy and N-hydroxy-2-acetylaminofluorene.

- 4289 BEHAVIOR OF OXIDATIVE AND HYDROLYTIC ENZYMES IN CADMIUM-INDUCED INTERSTITIAL CELL TUMORS IN ALBINO RATS. (Ger.) Knorre, D. (Regional Hosp. Psychiatry, Leipzig-Dosen, Germany). *Arch Geschwulstforsch* 42(2):127-134, 1973.

Determinations of succinate dehydrogenase, lactate dehydrogenase (LDH), and NADH₂-nitro-BT-reductase and lactate dehydrogenase isoenzymes were made on testicular sections from 152 male Wistar rats at various intervals after a single injection of cadmium chloride (550 µg/100 s.c.); 56 untreated rats served as controls. Enzyme activity had almost disappeared within 48 hr after injection when necrotic changes were observed. Reparative inflammation, which developed after 7-14 days, was associated with an increased activity of oxidative and hydrolytic enzymes in the interstitial tissue and fibrosis, which occurred in the peripheral part of the testes after one month, with a decrease in enzyme activity. Interstitial cell proliferation, which began after five months and became pronounced after 12 months, was accompanied by a slight increase in LDH and NADH₂-nitro-BT-reductase. Uni- or bilateral interstitial cell tumors which developed in 10 of the 152 rats 355-698 days after injection of cadmium chloride were strongly positive for LDH and NADH₂-nitro-BT-reductase, indicating that energy metabolism was increased. Succinate dehydrogenase activity was very low, suggesting that metabolism in the poorly differentiated tumor cells is primarily anaerobic. This was confirmed by increases in the muscle or M-fraction of LDH during tumor development.

- 4290 INFLUENCE OF ENVIRONMENT ON THE PHOTOOXIDATION OF TRYPTOPHAN WITH THE CARCINOGENIC HYDROCARBON 3,4-BENZOPYRENE IN AQUEOUS SOLUTIONS OF SOAP, CAFFEINE, AND UREA. (E.) Reske, G. (Inst. Phys. Biochem, U. Frankfurt/M, West Germany) and H. Bauer. *Z Naturforsch* 28C(7/8):390-396, 1973.

The solvent dependence of the photooxidation of tryptophan and 3,4-benzopyrene in aqueous solutions was studied by quantum yield measurements. When the hydrocarbon is dissolved in aqueous solution of caffeine, the quantum yields indicate a 3,4-benzopyrene photosensitized tryptophan oxidation instead of a photocooxidation, which is indicated in aqueous solution of sodium dodecylsulfate. The same photosensitized oxidation as in caffeine solution is observed, when urea (6 M) is added to the soap solution, while the fluorescence and absorption spectra indicate no change in the solvation state of the hydrocarbon, comparable to the change from hydrophobic solubilization by the detergent to dipole-induced dipole complex solubilization by caffeine.

It is concluded that the difference in the reaction pathways is caused by different solvation states of the excited or reacting oxygen.

- 4291 INDUCTION OF RAT MAMMARY TUMOURS IN ALTERED IODINE METABOLISM. (E.) Kellen, J. A. (Banting Inst., U. Toronto, Ontario, Canada). *Oncology* 28(3):269-273, 1973.

7,12-dimethylbenz(a)anthracene (DMBA) was injected i.v. into female Sprague-Dawley rats, some of whom were given drinking water containing small amounts of potassium iodide for 150 days following DMBA treatment, some of whom were maintained at 4°C for 50 days following DMBA treatment, and some of whom were given no further unusual treatment. The DMBA and DMBA + KI groups showed a 100% incidence of mammary tumor development by the end of the 150-day observation period. In contrast, 70% of the DMBA + cold-stressed animals developed tumors, while none of a group of nontreated animals developed tumors. Histology of the tumor nodules revealed variability from secretory adenomas to well-differentiated adenocarcinomas without preferential incidence of a certain type in any group. Thus, iodine appears to have a specific accelerating effect (possibly via the thyroid gland) on DMBA tumorigenesis.

- 4292 STUDY OF TOXINS ISOLATED FROM GRAIN INFECTED WITH *FUSARIUM SPOROTRICHIOIDES*. (E.) Akhmeteli, M. A. (Acad. Med. Sci., USSR, Moscow), A. B. Linnik, K. S. Cernov, V. M. Voronin, A. Ja. Hesina, N. A. Guseva and L. M. Sabad. *Pure Appl Chem* 35(3):209-215, 1973.

Experiments with mice showed that an aqueous extract of barley grain which had been attacked by *Fusarium sporotrichioides* is blastogenic. The mice were each given 0.5 ml of the extract through a gastric "sonde" five times/week for one year. During the year the mice were given about 125 ml of the extract. The extract caused a statistically significant increase in lung adenomas in general, and particularly in the males. Lung adenomas occurred in 4 of the 35 controls and in 18 of the 50 experimental animals. There was a significant association between tumor-bearing and alpha-fetoprotein when a sensitive immunoradiographic method was used.

- 4293 COMPARATIVE STUDIES WITH DIFFERENT DOSES OF *N*-NITROSOMORPHOLINE, *N*-NITROSOPIPERIDINE, *N*-NITROSOMETHYLUREA, AND DIMETHYLNITROSAMINE IN SYRIAN GOLDEN HAMSTERS. (E.) Haas, H. (Hannover Med. Sch., West Germany), U. Mohr and F. W. Krüger. *J Natl Cancer Inst* 51(4):1295-1301, 1973.

A total of 520 Syrian golden hamsters (*Mesocricetus auratus*) was treated s.c. with one-fifth, one-tenth, or one-twentieth LD50 *N*-nitrosomorpholine (NM), *N*-nitrosopiperidine (NP), *N*-nitrosomethylurea (NMU), or dimethylnitrosamine (DMN). The carcinogenicity of these compounds demonstrated selective organotropism. NM, as well as NP, resulted in 65-100% tracheal papillary tumors, dependent upon the applied dose

level; however, NM induced a higher incidence of tumors in the nasal cavities than did NP. A slight effect was observed in the lungs after treatment with NM. NMU induced up to 65% sarcomas localized at the site of injection, and in 7% of the animals papillomas of the forestomach were seen. With DMN application, overlapping hepatotoxic and carcinogenic effects were observed. Lower doses increased the survival rate, but tumor incidence was reduced only with the cyclic compounds.

- 4294 MAMMARY NODULES IN DOGS DURING FOUR YEARS' TREATMENT WITH MEGESTROL ACETATE OR CHLORMADINONE ACETATE. (E.) Nelson, L. W. (Mead Johnson Res. Ctr., Evansville, Ind.), J. H. Weikel, Jr. and F. E. Reno. *J Natl Cancer Inst* 51(4):1303-1311, 1973.

Mammary nodules were evaluated during the first 4 yr of a chronic toxicity study involving female beagle dogs treated for 7 yr with megestrol acetate or chlormadinone acetate. The evaluation included clinical observations and histopathologic examination of tissues from dogs killed at two and four years. Dosages were 1, 10, and 25 times the projected human dose of chlormadinone. Clinically, numerous palpable mammary nodules were found in dogs treated with middle and high doses of megestrol and with chlormadinone. Of 38 grossly detected nodules evaluated microscopically from the megestrol-treated dogs, 27 were nodular hyperplasias, five were benign mixed mammary tumors, three were ductal dilations, one was a lymph node, one was fat necrosis, and one was apparently the umbilicus. Twenty-two nodules from chlormadinone-treated dogs included 12 nodular hyperplasias, four benign mixed mammary tumors, three lymph nodes, one chondromucoid degeneration, and one adenocarcinoma; in one specimen, no nodule or mammary tissue was found. Involution, regression, and sclerosis of many areas of nodular hyperplasia were evident at 4 yr. These data suggest that the number of clinically palpable mammary nodules can be misleading and is not necessarily an index of neoplasia. Benign mixed mammary tumors and the one adenocarcinoma from the chlormadinone-treated dog appeared as distinct entities and were not related developmentally to nodular hyperplasia. The findings indicate that no projection of an adverse effect of progestogenic oral contraceptive steroids on the mammary tissue of the human female should be made without a more thorough understanding of such considerations as the natural causes of mammary changes of endocrinologic profiles, and duration of drug action in different test species.

- 4295 THE EFFECT OF PROLONGED DIETARY ADMINISTRATION OF THE NON-CARCINOGEN, 4-ACETYLAMINOFLUORENE ON THE LIVER OF THE RAT. AN ELECTRON MICROSCOPIC STUDY. (E.) Flaks, B. (U. Bristol Med. Sch., England). *Chem Biol Interact* 7(3):151-164, 1973.

The ultrastructural changes in the livers of male Leeds strain rats fed a diet containing 0.05% of the noncarcinogen 4-acetylaminofluorene (4-AAF) were studied. Liver cells from animals maintained

on this diet for up to 10 months showed progressive glycogen depletion and hypertrophy of the agranular endoplasmic reticulum. Several cells also showed enlargement of mitochondria and/or the presence of small cytoplasmic lipid inclusions. After six months of treatment, enlarged mitochondria were no longer apparent; however, increased numbers of lysosomes in the vicinity of the Golgi zones were seen. Although these effects were largely reversed following cessation of 4-AAF treatment, a small amount of glycogen depletion and cytoplasmic lipid inclusions as well as prominent agranular endoplasmic reticulum hypertrophy persisted. The failure of 4-AAF to produce granular endoplasmic reticulum lesions, cell surface changes, or persistent mitochondrial changes was strikingly different from the effects observed following treatment with the isomeric carcinogen 2-AAF.

- 4296 IDENTIFICATION AND PROPERTY OF THE MUTAGENIC PRINCIPLE FORMED FROM A FOOD-COMPONENT, METHYLGUANIDINE, AFTER NITROSATION IN SIMULATED GASTRIC JUICE. (E.) Endo, H. (Cancer Res. Inst., Kyushu U., Katakasu, Fukuoka, Japan) and K. Takahashi. *Biochem Biophys Res Commun* 54(4):1384-1392, 1973.

The active principle responsible for the mutagenicity of methylguanidine nitrosated in weakly acidic conditions and in simulated gastric juice was identified to be methylnitrosocyanamide (MNC). This compound was quite similar to N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) in its mutagenic quality, while its mutagenic activity was about 16 times higher for a strain of *Salmonella typhimurium* than that of the latter at neutral pH. MNNG (2.5 moles/ml) showed mutagenicity up to 160 fold dilution, while the same concentration of MNC was mutagenic up to 2560 fold dilution for the TA1535 strain. MNC and MNNG also showed mutagenic effects for TA1537 strain up to 256 fold and 16 fold dilution respectively but no activity for TA1536 nor TA1538 strains. It is therefore likely that MNC acts mutagenic not only as the base-pair substitution mutagen but also the frameshift mutagen as MNNG.

- 4297 THE FORMATION OF CARCINOGENIC NITROSO COMPOUNDS FROM NITRITE AND SOME TYPES OF AGRICULTURAL CHEMICALS. (E.) Elespuru, R. K. (U. Tennessee-Oak Ridge Grad. Sch. Biomed. Sci.) and W. Ijinsky. *Food Cosmet Toxicol* 11(5):807-817, 1973.

Many widely used agricultural chemicals are derivatives of alkylureas and alkylcarbamic acids. Since these compounds have the potential to react with nitrite in acid solution to form N-nitroso compounds, many of which are carcinogenic, the reactions of some dialkylureas and N-alkylcarbamates with nitrous acid were examined. The interaction of trisubstituted ureas with nitrite in mildly acid solution can lead to two products, the appropriate nitrosourea and, by nitrosative cleavage, the dialkylnitrosamine. Except with N-phenyldialkylureas, the nitrosourea is the major product. Phenyldialkylureas and trisubstituted thioureas give dialkylnitrosamine as the sole N-nitroso product. Tetraalkylureas and bis-(dialkyl-

thiocarbamyl)-disulphides also form dialkylnitrosamines by reaction with nitrous acid. The yields of these carcinogenic nitrosamines are significant at 37°C and at low concentrations of the tertiary amine (0:01 M). The insecticide, carbaryl, reacts with nitrous acid to form a highly mutagenic N-nitroso derivative. Since nitrites are present in many foods, a carcinogenic hazard to man could exist when food containing residues of certain agricultural chemicals is simultaneously present in the stomach. Most of these chemicals have been tested by long-term feeding to experimental animals but, to the authors' knowledge, none of them has yet been fed together with nitrite.

- 4298 LACK OF CARCINOGENIC ACTION FOR CYCLAMATE, CYCLOHEXYLAMINE AND SACCHARINE IN RATS. (Ger.) Schmahl, D. (German Cancer Res. Inst., Heidelberg). *Arzneim Forsch* 23(10):1466-1470, 1973.

An attempt was made to confirm the results of American investigators who found that artificial sweeteners produced bladder cancer in experimental animals. Seven groups of Sprague-Dawley rats, each consisting of 52 males and 52 females, were fed cyclamate (total doses of 882 or 2188 g/kg); saccharine (totals of 83 or 210 g/kg); cyclamate (totals of 750 or 1852 g/kg) + saccharine (totals of 75 or 185 g/kg); or cyclohexylamine (total of 177 g/kg). An untreated group of rats served as controls. The strain of rats employed had a high incidence of spontaneous tumors and developed bladder cancer after administration of butylbutanol nitrosamine. All rats were followed for their entire life span. Only one rat treated with 2% cyclamate developed a solitary bladder papilloma after 798 days. The bladder mucosa was normal in more than 95% of the rats. Parasites were present in the bladders of 16% of all cases and 8 older rats had bladder stones. None of the sweeteners tested had any effect on weight gain, blood picture, systolic blood pressure, morphology of the heart or bladder, survival time, or the incidence of any type of tumor. Despite its catecholamine-like action, cyclohexylamine had no effect on blood pressure and caused no morphological damage to the cardiovascular system.

- 4299 RELATIONSHIP OF DIETARY SELENIUM CONCENTRATION; CHEMICAL CANCER INDUCTION; AND TISSUE CONCENTRATION OF SELENIUM IN RATS. (E.) Harr, J. R. (Dept. Vet. Med., Oregon State U., Corvallis). *Clin Toxicol* 6(3):487-495, 1973.

The effect of dietary selenium on carcinogenesis was studied in selenium-depleted, vitamin E-supplemented female OSU-brown rats fed a ration containing the known carcinogen N-2-fluorenyl acetamide (FAA). Addition of dietary selenium (0.1, 0.5 or 2.5 ppm) resulted in an 18-fold increase in liver selenium (0.40 to 7.35 ppm) but had no effect on muscle selenium content when compared with controls. Liver selenium concentration was inversely related to the incidence of FAA-induced mammary and hepatic cancer and to early mortality. After 211 days, 38 of 40 rats fed a high selenium diet (1.5 or 2.5 ppm) were alive without evidence of s.c.

neoplasm, whereas 24 of the 40 rats fed a low selenium diet (0.1 ppm) had evidence of neoplasm.

4300 STIMULATION OF CORTISONE AND RADIORESISTANT MOUSE LYMPHOID CELLS *IN VITRO* BY PHYTO-HAEMAGGLUTININ-P (PHA), POKEWEE MITOGEN (PWM) AND CONCAVALIN A (CON A). (Ger.) Rühl, H. (Steglitz Clin., Free U. Berlin, Germany), W. Vogt, U. Rühl, G. Bochart and S. Schmidt. *Klin Wochenschr* 51(20):1026-1027, 1973.

4301 BIFUNCTIONAL PHOTOBINDING OF PSORALEN TO SINGLE STRANDED NUCLEIC ACID. (E.) Marciani, S. (Inst. Pharmaceutical Chem., Padua U., Padova, Italy), M. Terbojevich, F. Dall'Acqua and G. Rodighiero. *Z Naturforsch* 28(7-8):370-375, 1973.

4302 METHYLCHOLANTHRENE CARCINOGENESIS IN THE NORTH AMERICAN OPOSSUM (*DIDELPHIS VIRGINIANA*). (E.) Andrews, E. J. (Coll. Med., Pennsylvania St. U., Hershey). *J Natl Cancer Inst* 51(4):1217-1225, 1973.

4303 EFFECT OF STREPTOZOTOCIN IN THE CHINESE HAMSTER (*CRICETULUS GRISEUS*). (E.) Berman, L. D. (Boston City Hosp., Mass.), J. A. Hayes and T. M. Sibay. *J Natl Cancer Inst* 51(4):1287-1294, 1973.

4304 CARCINOGENICITY OF PROPOSED CANCER CHEMOTHERAPEUTIC AGENTS WITH STILBENE-ARYLNI-TROSAMINE STRUCTURES. (E.) Yamamoto, R. S. (Natl. Inst. Hlth., Bethesda, Md.), H. L. Richardson, E. K. Weisburger, J. H. Weisburger, T. Benjamin and C. T. Bahner. *J Natl Cancer Inst* 51(4):1313-1315, 1973.

4305 INDUCTION OF TUMORS IN RATS BY ORAL ADMINISTRATION OF TECHNICAL ACID VIOLET 6B. (E.) Uematsu, K. (Hyogo Coll. Med., Osaka, Japan) and T. Miyaji. *J Natl Cancer Inst* 51(4):1337-1338, 1973.

4306 PATHOLOGIC EFFECTS IN RATS SURVIVING PRE-NATAL AND NEONATAL ADMINISTRATION OF 1,3-BIS(2-CHLOROETHYL)-1-NITROSOUREA. (E.) Weiss, E. deC. (New York U. Med. Ctr., N.Y.), H. Cravioto, J. F. Weiss and J. Ransohoff. *J Natl Cancer Inst* 51(4):1363-1365, 1973.

4307 GENETIC CHARACTERIZATION OF *AD-3* MUTANTS INDUCED BY CHEMICAL CARCINOGENS, 1-PHENYL-3-MONOMETHYLTRIAZENE AND 1-PHENYL-3,3-DIMETHYLTRIAZENE, IN *NEUROSPORA CRASSA*. (E.) Ong, T.-M. (Natl. Inst. Environmental Hlth. Sci., Res., Triangle Park, N.C.) and F. J. De Serres. *Mutat Res* 20(1):17-23, 1973.

4308 THE ASSOCIATION BETWEEN PURINE NUCLEOSIDES AND BENZENE IN AQUEOUS SOLUTION, STUDIED BY PROTON MAGNETIC RESONANCE AND NUCLEAR OVERHAUSER ENHANCEMENTS. (E.) Lüdemann, H.-D. (Dept. Biol., U. Regensburg, Germany) and E. v. Goldammer. *Z Naturforsch* 28(7-8):361-369, 1973.

4309 INCREASED AFLATOXIN PRODUCTION BY *ASPERGILLUS FLAVUS* VIA COBALT IRRADIATION. (E.) Applegate, K. L. (Dept. Poultry Sci., Ohio St. U., Columbus) and J. R. Chipley. *Poult Sci* 52(4):1492-1496, 1973.

4310 ELECTRONIC SPECTRA AND ELECTRONIC STRUCTURES OF AMINOANTHRACENES. (E.) Schulman, S. G. (Coll. Pharmacy, U. Florida, Gainesville), P. J. Kovi, G. Torosian, H. McVeigh and D. Carter. *J Pharm Sci* 62(11):1823-1826, 1973.

4311 LEAD CONCENTRATION IN HUMAN BRAIN TISSUE. AN AUTOPSY STUDY. (E.) Zaworski, R. E. (Northwestern U. Med. Sch., Chicago, Ill.) and R. Oyasu. *Arch Environ Health* 27(6):383-386, 1973.

4312 *IN VIVO* TRANSPLANTABILITY OF URINARY BLADDER EPITHELIA FROM RATS INGESTING *N*-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]FORMAMIDE. (E.) Yalciner, S. (St. Vincent Hosp., Worcester, Mass.), A. J. Tiltman and G. H. Friedell. *J Natl Cancer Inst* 51(4):1177-1185, 1973.

4313 BENIGN HEPATOMAS AND ORAL CONTRACEPTIVES. (E.) Contostavlos, D. L. (Office Med. Examiner, Philadelphia, Pa.). *Lancet* (7839):1200, 1973.

4314 ENHANCEMENT OF PLATELET AND MUSCLE ACTOMYOSIN ATPase ACTIVITY AND SUPERPRECIPITATION BY THE TUMOUR-PROMOTER 12-O-TETRADECANOYL-PHORBOL-13-ACETATE (PMA). (E.) Puszkun, E. G. (Mount Sinai Sch. Med., City U. New York, N.Y.) and M. B. Zucker. *Nature [New Biol]* 245(148):277-280, 1973.

4315 BREAST CANCER ASSOCIATED WITH REPEATED FLUOROSCOPY. (E.) Grundy, G. W. (Natl. Inst. Hlth., Bethesda, Md.) and B. G. Uzman. *J Natl Cancer Inst* 51(4):1339-1340, 1973.

4316 INTERACTION OF CYTOCHROME OXIDASES *AA₃* AND *D* WITH NITRITE. (E.) Meyer, D. J. (Dartmouth Med. Sch., Hanover, N.H.). *Nature [New Biol]* 245(148):276-277, 1973.

4317 CHANGES IN PRODUCTION OF GONADOTROPIC HORMONES AND ANDROGENS IN EXPERIMENTAL REPRODUCTION OF TUMOURS OF THE TESTES. (Rus.) Dmitriev, V. N. (Izhevsk Med. Inst. USSR). *Biull Eksp Biol Med* 76(8):100-102, 1973.

- 4318 ULTRASTRUCTURE AND REVERSIBILITY OF BLADDER CARCINOMA OF RATS PRODUCED BY FEEDING OF N-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]FORMAMIDE. (E.) Pai, S. H. (Methodist Hosp. Brooklyn, New York, N.Y.), L. Amaral, S. Werthamer and F. G. Zak. *Invest Urol* 11(2):125-135, 1973.
- 4319 TERATOGENIC EFFECTS OF CARCINOGEN IMPLANTATION IN A REGENERATIVE FIELD IN *BUFO ARENARUM* TADPOLES. (E.) Matos, E. L. (Angel H. Roffo Oncology Inst., Buenos Aires, Argentina) and E. S. De Lustig. *Teratology* 8(2):167-173, 1973.
- 4320 EFFECT OF COBALT ON INDUCED CARCINOGENESIS OF THE SKIN. (E.) Finogenova, M. A. (Lab. Carcinogens, USSR Acad. Med. Sci., Moscow). *Bull Exp Biol Med* 75(2):168-169, 1973.
- 4321 EFFECT OF DIMETHYLNITROSAMINE ON MOUSE EMBRYONIC KIDNEY TISSUE IN ORGAN CULTURE. (E.) Bogovskii, S. P. (Inst. Exp. Clin. Oncology, USSR Acad. Med. Sci., Moscow) and Y. D. Sorokina. *Bull Exp Biol Med* 75(2):170-172, 1973.
- 4322 RENAL PELVIC CARCINOMA AFTER THOROTRAST PYELOGRAPHY. CASE REPORT. (E.) Westin, J. (Sahlgren's Hosp., U. Göteborg, Sweden), L.-O. Lanner, A. Weinfeld and L. Zettergren. *Acta Med Scand* 194(1-2):141-143, 1973.
- 4323 ACTION MECHANISM OF N-NITROSO-N-ALKYLUREAS ON MONONUCLEOTIDES. STUDY OF THE REACTION OF ADENOSINE-5'-MONOPHOSPHATE WITH NITROSOMETHYLUREA. (Rus.) Bedniak, A. E. (Khabarovsk State Med. Inst., USSR) and V. G. Gus'kov. *Dokl Akad Nauk SSSR* 212(6):1451-1454, 1973.
- 4324 THE EFFECTS OF CHROMIUM COMPOUNDS ON THE HUMAN BODY. (Jap.) Inoue, K. (Hokkaido Hyg. Res. Inst., Japan), T. Ishihara, R. Nakayama and R. Kotani. *J Pollution Control* 9(5):457-460, 1973.
- 4325 BIOLOGICAL ANTIOXIDANTS AS REGULATORS OF OXIDATIVE FREE RADICAL PROCESSES IN THE EARLY STAGES OF HEPATOCARCINOGENESIS. (Ukr.) Sydoryk, Ye. P. (Inst. Oncol. Problems, Kiev, USSR), T. M. Iurkivs'ka and Ye. A. Baglei. *Ukr Biokhim Zh* 44(3):365-368, 1972.
- 4326 PHENACETIN AND URINARY TRACT TUMORS. (Fr.) Wahlqvist (Salgrenska Hosp., Gothenburg, Sweden). *J Urol Nephrol* 78(12 bis):234-235, 1972.
- 4327 SPECTROSCOPIC INTERACTION OF AFLATOXIN B₁ AND A METABOLITE WITH HEPATIC MICROSOMES. (E.) Gurtoo, H. L. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Proc Am Assoc Cancer Res* 14(March):29, 1973.
- 4328 CUTANEOUS ARSENICALISM WITH A LONG LATENT PERIOD. ARSENICAL CANCERS. (Fr.) L'Epee, P. (Inst. Industrial Med., U. Bordeaux II, France), L. Texier, H. J. Lazarini, G. Ducombs, J. Doignon, S. Larcebau and M. J. Miegville. *Arch Mal Prof* 34(7/8):457-461, 1973.
- 4329 ACUTE LEUKEMIA FOLLOWING CHLORAMPHENICOL EXPOSURE? THREE CASE REPORTS AND REVIEW OF THE LITERATURE. (Ger.) Gadner, H. (Pediatr. Clin., Free U., Berlin, Germany), U. Gethmann, K. Jessenberger and H. Riehm. *Monatsschr Kinderheilk* 121(9):590-594, 1973.
- 4330 THE MYCOFLORA AND AFLATOXIN B₁ CONTENT OF PEANUT SAMPLES. (Ger.) Gemeinhardt, H. (Hyg. Inst., Berlin, Germany) and G. Krug. *Zentralbl Bakteriol* 128(1/2):42-50, 1973.
- 4331 EXPERIMENTAL STUDIES ON THE CARCINOGENICITY AND RETENTION OF BENZO(a)PYRENE AT THE SITE OF ADMINISTRATION AFTER INTRATRACHEAL ADMINISTRATION AND SUBCUTANEOUS INJECTION. (Ger.) Pott, F. (Med. Inst. Air Hyg. Silicosis Res., U. Düsseldorf, Germany), R. Tomingas and F. J. Reiffer. *Zentralbl Bakteriol Hyg [Orig B]* 158(2):97-108, 1973.
- 4332 SPONTANEOUS TUMORS IN SYRIAN GOLDEN HAMSTERS. (Ger.) Dontenwill, W. (Cigarette Industry Res. Inst., Hamburg, Germany), H.-J. Chevalier, H. P. Harke, U. Lafrenz and G. Reckzeh. *Z Krebsforsch* 80(2):127-158, 1973.
- 4333 EVALUATING OF THE ALKYLATING POTENTIAL OF ONCOLOGICALLY IMPORTANT PHOSPHORIC ACID ESTERS WITH THE 4-(4-NITROBENZYL)PYRIDINE REACTION. (Ger.) Fischer, G. W. (Res. Inst. Chem. Toxicol., Leipzig, Germany) and K. Lohs. *Arch Geschwulstforsch* 42(1):34-40, 1973.
- 4334 PHENACETIN ABUSE AND TUMORS OF THE URINARY TRACT. (Fr.) Rathert, P. (Fac. Med., Rhine-Westphalia Technol. Coll., Aachen, Germany), H. Melchior and W. Lutzeyer. *J Urol Nephrol* 78(12 bis):230-234, 1972.
- 4335 ARYL HYDROCARBON HYDROXYLASE INDUCTION IN MAMMALIAN LIVER CELL CULTURE. IV. STIMULATION OF THE ENZYME ACTIVITY IN ESTABLISHED CELL LINES DERIVED FROM RAT OR MOUSE HEPATOMA AND FROM NORMAL RAT LIVER. (E.) Benedict, W. F. (Natl. Inst. Child Hlth. Human Development, Bethesda, Md.). *Biochem Pharmacol* 22(21):2766-2769, 1973.
- 4336 AUTORADIOGRAPHIC STUDIES OF THE DNA SYNTHESIS IN LYMPHOCYTES OF THE SPLEEN AND LYMPH NODES IN MICE WITH TUMOR GROWTH. (Rus.) Gudim-Levkovich, K. A. (Inst. Oncological Problems, Ukrainian SSR Acad. Sci., Kiev). *Vopr Onkol* 19(9):75-79, 1973.

4337 HISTOLOGICAL AND AUTORADIOGRAPHIC STUDIES OF HUMAN ENDOMETRIUM DURING SEQUENTIAL THERAPY WITH MESTRANOL AND LYNESTRENOL. (Ger.) Kaltenbach, F. J. (Clinic Gynecol. Obstet., U. Freiburg im Breisgau, Germany), O. Fettig and J. Welter. *Arch Gynaekol* 215(3):325-342, 1973.

4338 PERIPHERAL NERVE TUMORS INDUCED IN HAMSTERS. (Georg.) Beniashvili, D. Sh. (No affiliation). *Soobshch Akad Nauk Gruzinsk SSR* 71(2):489-492, 1973.

4339 CARCINOGENIC HAZARDS IN THE PRODUCTION OF CHROMIUM FERROALLOYS. (Rus.) Pokrovskaya, L. V. (Inst. Industrial Hyg. Occupational Hlth., Sverdlovsk, USSR) and N. K. Shabynina. *Gig Tr Prof Zabol* (10):23-26, 1973.

4340 ONCOGENIC PROPERTIES OF PRODUCTS OBTAINED BY CHLORINATION AND OZONIZATION OF BENZO(a)PYRENE. (Rus.) Il'nitskii, A. P. (Inst. Exp. Clin. Oncol., Moscow, USSR) and V. M. Voronin. *Gig Sanit* 9(1):22-25, 1973.

4341 KINETICS OF PHOTOINDUCED DEGRADATION OF SOME METHYL DERIVATIVES OF BENZO(a)PYRENE. (Rus.) Gubergrits, M. (Inst. Chem., Tartu, USSR), L. Paalme and J. Pahapill. *Izv Akad Nauk Estonsk SSR Khim Geol* 22(1):31-36, 1973.

4342 ADMINISTRATION OF ³H-BENZO(a)PYRENE TO PREGNANT MICE AND RATS: INCORPORATION OF RADIOACTIVITY INTO SUBCELLULAR FRACTIONS OF VARIOUS TISSUES. (Ger.) Wunderlich, V. (Central Inst. Cancer Res., Berlin-Buch, Germany) and I. Tetzlaff. *Arch Geschwulstforsch* 42(2):95-106, 1973.

4343 RAPID UPTAKE OF DIETARY CHOLESTEROL BY HYPERPLASTIC LIVER NODULES AND PRIMARY HEPATOMAS. (E.) Horton, B. J. (McArdle Lab., U. Wisconsin, Madison), G. E. Mott, H. C. Pitot and S. G. Goldfarb. *Proc Am Assoc Cancer Res* 14(March):78, 1973.

4344 THE RELATIONSHIP BETWEEN TISSUE TOXICITY AND THYMOMA INDUCTION IN INBRED SWISS MICE BY ALKYLNITROSAMIDES. (E.) Maitra, S. C. (Dept. Path., U. Western Ontario, London, Canada) and J. V. Frei. *Proc Am Assoc Cancer Res* 14(March):81, 1973.

See also:

- * (Rev): 4202, 4203, 4240, 4244, 4248, 4253, 4255, 4256
- * (Phys): 4347
- * (Viral): 4355, 4407
- * (Immun): 4467, 4475, 4485
- * (Path): 4517
- * (Epid-Biom): 4561, 4577, 4590, 4591

- 4345 DEMONSTRATION OF AN ELECTRONIC CHANGE DURING CARCINOGENESIS. (Fr.) Marmasse, C. (Fac. Sci., Free Natl. U. Mexico, Mexico City). *C R Acad Sci (Paris)* [D] 276(24):3215-3217, 1973.

Redox potentials were measured on cell-free liver homogenates from 3 groups of fasted Wistar rats which were fed: (1) a low 8% protein diet and butter yellow (dose unspecified) for several months, (2) a low 8% protein diet or (3) a normal diet. A trace of methylene blue and a little n-octanol were added to each tube after dilution of homogenates with phosphate buffer. After oxygen had been removed from the homogenates by bubbling pure argon through them, measurements were made with gold electrodes against a saturated calomel electrode at 37.8 ± 0.1 C. Statistical analysis of the results showed that the mean redox potential was significantly increased in homogenates from rats with experimental hepatomas. The mean redox potential of homogenates from the tumor nodule itself was much higher than that of homogenates from other parts of the liver. Since the redox potential of liver homogenates increased in the early stages of carcinogenesis before any detectable change had occurred in biochemical activity or histology, slight but detectable electrical activity may result in a change in at least one system of electron transfer during the early stages of carcinogenesis.

- 4346 EFFECTS OF ^{60}Co GAMMA RADIATION ON THE DISTRIBUTION, CATABOLISM, AND INCORPORATION OF ^{125}I -5-iodo-2'-deoxyuridine INTO INTESTINAL AND SPLENIC DEOXYRIBONUCLEIC ACID IN MICE. (E.) Johanson, K. J. (Dept. Radiation Biol., Gustaf Werner Inst., Sweden). *Acta Radiol (Ther)* (Stockh) 11(6): 556-564, 1972.

The effect of 200 rad ^{60}Co gamma radiation on the kinetics of the distribution and catabolism of ^{125}I -5-iodo-2'-deoxyuridine ($^{125}\text{IUdR}$) was investigated in male NMRI mice weighing 26 to 28 g. The mice were given water ad libitum but had no access to food from 20 hr before irradiation. Ten μCi $^{125}\text{IUdR}$ and 0.1 μ mole carrier IUdR were injected i.p. 2 hr after irradiation or sham irradiation. No significant changes were observed in the irradiated mice compared with the control mice. The small differences that were seen might be explained by the decreased incorporation of $^{125}\text{IUdR}$ into DNA in the irradiated mice. Fluorouracil (5×10^{-5} mole) injected i.p. 10 min before the $^{125}\text{IUdR}$ injection increased its incorporation into DNA. This increase was more marked at 2 hr after irradiation, indicating the changes in the thymidine pool due to flooding of thymidine from dead cells is of minor importance.

- 4347 INHIBITORY EFFECT OF CAFFEINE ON THE INDUCTION OF SKIN CANCER IN MICE BY ULTRAVIOLET RADIATION. (Fr.) Zajdela, F. (Curie Fdn., Radium Inst., Paris, France) and R. Latarjet. *C R Acad Sci (Paris)* [D] 277(12):1073-1076, 1973.

Female Swiss (Carsharlton) mice were exposed to UV radiation from a mercury vapor lamp for 90 min 5

times a week for 27 weeks (total dose of 1×10^7 ergs/mm²). Before each exposure, the right ears of these mice were painted with 0.2% caffeine dissolved in acetone and chloroform (1:1) while the left ears were untreated. Malignant tumors (90% keratogenic epitheliomas and 10% subepidermal sarcomas) developed on 84-89% of the untreated ears 5-11 months after irradiation was begun. Treatment with caffeine reduced the incidence of tumors to 54% in one experiment and to 47% in another. The difference in the incidence of tumors on treated and untreated ears was highly significant. Caffeine inhibited the development of tumors to the same extent as was previously found for theophylline. To rule out the possibility that caffeine and theophylline might act as screening agents, their tumor inhibiting effect was compared with that of uridine in an alcohol-acetone solution. Uridine did not decrease the incidence of tumors (93%). Since caffeine inhibits the repair of photo-induced lesions in nucleic acids, these findings suggest that cancers induced by UV radiation result from DNA repair which permits the cell to survive but allows a particular error to remain in this DNA.

- 4348 PROLIFERATIVE AND NEOPLASTIC CHANGES IN THE OVARIES OF HAMSTERS TREATED WITH ^{131}I -IODINE AND METHYLTHIOURACIL. (E.) Christov, K. (Cancer Res. Inst., Sofia, Bulgaria) and R. Raichev. *Neoplasma* 20(5):511-516, 1973.

The appearance and structure of the ovarian tumors in hamsters treated with ^{131}I (10 μCi , i.p.) and methylthiouracil (20 mg/day) alone or in combination were studied. Nodular cell proliferation and tumor growth were observed in 9 of the 10 ^{131}I -iodine injected animals killed between the 600th and 620th day of the experiment. The induced tumors were mainly of granulosa and granulosa-theca cell type. Diffuse hyperplasia of the ovarian mesenchymal-stromal cells in methylthiouracil and in combined ^{131}I -iodine + methylthiouracil treatment was noted. The histogenesis of the ovarian tumors and the effect of ^{131}I -iodine and methylthiouracil on the ovarian carcinogenesis in hamsters are discussed. It is suggested that ^{131}I damages the thyroid epithelia, causing hyperplasia and proliferation of thyroid stimulating hormone (TSH) and chromophobic pituitary cells. It was previously shown that part of the chromophobic cells become thyrotrophins, part become gonadotrophins. It is further suggested that radioactive iodine may induce small changes in the ovarian cells, leading to eventual tumor development under increased TSH stimulation.

- 4349 UNIFORMITY OF RADIATION-INDUCED MUTATION RATES AMONG DIFFERENT SPECIES. (E.) Abrahamson, S. (Dept. Zoology, U. Wisconsin, Madison), M. A. Bender, A. D. Conger and S. Wolff. *Nature* 245(5428):460-462, 1973.

Data for radiation-induced forward mutation rates were reexamined for organisms as disparate as bacteria, fungi, higher plants, insects, and mammals

for which the amounts of DNA/nucleus were also known. The mutation rates/locus/rad varied over three orders of magnitude. When these rates were adjusted for the amount of DNA/nucleus and thus normalized to a common biological baseline, the mutation rates obtained were all essentially the same, varying by a factor of three instead of 1,000. For all species investigated, the unweighted average of the mutation rates normalized to the human DNA value is 2.6×10^{-7} /locus/rad. The consistency that obtains when the data are adjusted for the amount of DNA/nucleus for each species indicates either that it is the nucleus, and not the locus, that determines target size, or that, on the average, the size of a (radiation-mutable) locus is proportional to the total genome size (DNA content) for the species. This correspondence allows one to extrapolate from mutation rates obtained in experimental organisms to man with greater confidence.

- 4350 ROLE OF THE DIRECT ACTION OF RADIATION IN THE MECHANISM OF DEVELOPMENT OF MAMMARY GLAND TUMORS. (E.) Moskalev, Yu. I. (Inst. Biophys., Moscow, USSR). *Bull Exp Biol Med* 75(5): 546-548, 1973.

The anterior half of the body of 151 female non-inbred albino rats was irradiated in one series of experiments and the posterior half of the body was irradiated in a second series of 233 rats of both sexes. The dose rate was 22 R/min. The doses used ranged from 200 to 1000 rad. Mammary gland tumors appeared considerably earlier and in larger percentages in irradiated rats than in controls. In irradiated rats the tumors developed almost entirely in the irradiated glands whereas in controls the tumors developed in anterior or posterior sections with equal frequency. Mammary gland tumors, which develop spontaneously in males only rarely, did develop in males after irradiation of the posterior half of the body in doses of 400-800 rad. Throughout the entire observation period mammary gland tumors in males were always found only in the irradiated half of the body. After 16.7 months, the frequency of mammary gland tumors in males was 10% and in females was 56.8%. In females the incidence of mammary gland tumors increased regardless of whatever dose of radiation was used. Mammary gland tumors did develop sooner in females on exposure to X-rays.

- 4351 THE BINDING OF POLONIUM-210 TO RAT TISSUES. (E.) Lanzola, E. E. (Hygiene Inst., U. Pavia, Italy), M. E. Allegrini and D. M. Taylor. *Radiat Res* 56(2):370-384, 1973.

- 4352 EFFECT OF LOCAL RADIOTHERAPY ON CONSECUTIVE PRIMARY CANCERS IN PATIENTS WITH CARCINOMA OF THE BREAST. (E.) McCredie, J. A. (Dept. Surg., U. Western Ontario, London, Canada), W. R. Inch and M. Alderson. *Vth Perugia Quadrennial Intl Conference on Cancer* 122, 1973.

- 4353 MULTIPLE PRIMARY MALIGNANCY AND RADIATION EXPOSURE: HIROSHIMA AND NAGASAKI. (E.) Steer, A. (Atomic Bomb Casualty Commission, Hiroshima, Japan) and T. Wakabayashi. *Vth Perugia Quadrennial Intl Conference on Cancer* 16, 1973.

See also:

- * (Rev): 4208
- * (Chem): 4300
- * (Viral): 4367, 4397
- * (Immun): 4490
- * (Epid-Biom): 4549, 4580

- 4354 DIFFERENCES IN CYTOCHALASIN B INHIBITION OF 3-O-METHYLGLUCOSE UPTAKE BETWEEN BALB/3T3 CELLS AND A MURINE SARCOMA VIRUS TRANSFORMED CLONE. (E.) Graff, J. C. (Flow lab., Inc., Rockville, Md.), D. J. Hanson and M. Hatanaka. *Int J Cancer* 12(3): 602-612, 1973.

The effect of the mold metabolite cytochalasin B, on 3-O-methylglucose uptake was studied in cultured normal and murine sarcoma virus-transformed BALB/3T3 cells using isotopically labeled substrates. Cytochalasin B (10 µg/ml) inhibited the initial rapid rate of 3-O-methylglucose uptake in both normal and transformed cells. After this initial inhibition, transformed cells, but not normal control cells, continued to take up 3-O-methylglucose at a significant rate. This difference between control and transformed cells occurred despite an almost complete inhibition by cytochalasin B of 2-deoxyglucose uptake. Cytochalasin B had no effect on 3-O-methylglucose metabolism once it entered the cell. After 10 min, when intracellular 3-O-methylglucose concentrations were approaching saturation, uptake was still greater in transformed cells. These differences in 3-O-methylglucose uptake were maintained over a 10,000-fold concentration range of cytochalasin B. 3-O-Methylglucose uptake by transformed cells was found to be both concentration- and temperature-dependent. Loss of the initial rapid phase of uptake at lower temperatures coupled with a lack of cellular ability to concentrate the substrate and an accelerated substrate efflux rate in preloaded cells indicated that the transport process is controlled by a carrier-mediated facilitated diffusion system.

- 4355 ACCELERATED CLINICAL COURSE OF MAREK'S DISEASE IN CHICKENS FED PENICILLIN. (E.) Morris, J. A. (Food Drug Admin., Rockville, Md.), C. G. Aulisio, B. Mason, C. W. Shaw and R. C. Reisinger. *Proc Soc Exp Biol Med* 144(2):478-482, 1973.

The clinical course of Marek's disease was studied in white Leghorn chickens which received oral antibiotic in their drinking water prior to and following i.p. inoculation with the JM strain of Marek's disease virus (MDV). All chickens were bred and maintained until the time of the experiment under specific pathogen-free conditions. Whereas no deaths occurred among control animals during the first 23 days after MDV inoculation, six of 12 animals receiving oral penicillin died (250 µg/ml of drinking water/day). Only one death was observed among 56 chickens receiving one of five other antibiotics. Although deaths occurred in all groups between days 23 and 30, the number in the penicillin (8 of 12) and streptomycin (6 of 12) treated groups was significantly higher than any other group. These differences were clearly observable as late as 42 days after MDV inoculation. A greatly increased death rate was also observed in penicillin-supplemented chickens previously exposed to MDV by contact infection and in penicillin-supplemented chickens maintained in an MDV-contaminated environment. The death rate of streptomycin-supplemented chickens maintained in an MDV-contaminated environment was not enhanced.

- 4356 LOCALIZATION OF VIRAL ANTIGENS, DETERMINED BY IMMUNOPEROXIDASE, IN MAMMARY ADENOCARCINOMA IN MICE. (Fr.) Thomas, J. A. (Lab. Cell Biol., U. Paris, France), S. Garzon and C. Lambre. *C R Acad Sci [D] (Paris)* 277(4):457-462, 1973.

Immunoperoxidase staining was used to investigate viral antigens in three strains of mouse mammary adenocarcinoma. These strains were obtained by: (1) passage of the T III strain in Swiss mice and the consanguineous LAB subline, (2) passage of the T III strain *in vitro*, and (3) and reinoculation of the *in vitro* T III strain into LAB mice. Tumor slices induced by strain (1) were initially peroxidase-positive, but became peroxidase-negative starting with the 52nd passage when no A or B particles were present; these cells have remained negative up to the 91st passage. Cells infected with strain (2) contained no A or B particles and were peroxidase-negative between the 359th and 375th passages. Tumors induced with strain (3) were very positive: peroxidase was present in the cytoplasm, on apical cell membranes, and inside the galactophoric canals. The location of peroxidase-positive structures and their number in the cells corresponded to the location and number of A and B particles of mouse mammary tumor virus.

- 4357 STIMULATION OF PROTEIN SYNTHESIS AND OF RNA POLYMERASE ACTIVITY IN THE LIVER OF 3-METHYLCHOLANTHRENE-TREATED RATS. (It.) Greco, C. (Queen Elena Inst. Tumor Res., Rome, Italy) and U. Ferrini. *Tumori* 59(2):119-127, 1973.

Hydroxylation of N-2-fluorenylacetamide (FAA) and RNA polymerase activity were investigated in subcellular fractions from liver homogenates from male Wistar rats sacrificed at various intervals after a single i.p. injection of 1 mg of 3-methylcholanthrene (MCA) dissolved in 0.5 ml of seed oil. FAA hydroxylase activity increased rapidly in the microsomal fraction, doubling in the first 2 hr and reaching values five times those present initially 6 hr after treatment. On sedimentation in sucrose gradients, a significant increase occurred in the more aggregated fractions and a corresponding decrease in oligomeric fractions of polyribosomes isolated from rat liver 16 hr after administration of MCA. The activities of Mg^{++} - and $Mn/(NH_4)_2SO_4$ -dependent RNA polymerases increased in isolated nuclei, particularly in the chromatin of the extranucleolar fraction after injection of MCA. These findings suggest that MCA and its metabolites selectively regulate the rate of RNA synthesis and transport of messenger RNA into the cytoplasm.

- 4358 ACTIVATION OF ROUS SARCOMA VIRUS REPLICATION IN NATURALLY RESISTANT CULTURES OF HUMAN EMBRYONIC CELLS. (Rus.) Kuznetsov, O. K. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR), A. I. Zhudina and A. M. Diad'kova. *Vopr Onkol* 19(4):40-46, 1973.

Active Rous sarcoma virus (RSV, strain D-5), grown in chick embryonic cultures, was concentrated and

inoculated into monolayers of human embryonic fibroblasts. At 37 C, RSV was completely inactivated within 18-24 hr after inoculation; no replication or transformation were observed. Concentrated Sendai virus (strain 390), grown in the allantoic cavity of chick embryos, was 99.9% inactivated by exposure to UV radiation for 10 min. Morphological signs of cell transformation were detected in some cultures of human embryonic fibroblasts treated with active RSV and inactivated Sendai virus. RSV in these transformed cultures induced sarcomas in chicks; it also replicated and caused transformation in chick embryonic fibroblasts. If Sendai virus was added to the cultures before RSV, RSV did not replicate. This could be accounted for if the Sendai virus produced interferon which inactivated RSV. When RSV was added before Sendai virus, replication of RSV decreased as the interval between addition of the viruses increased. Replication of RSV was most pronounced when RSV and Sendai virus were added to the cultures simultaneously. The optimal dose of RSV was $10^{7.2}$ focus-forming units/0.5 ml, and the optimal dose of Sendai virus was 10,000-40,000 hemagglutinating units/0.5 ml. It is suggested that Sendai virus may have a two-fold action: it may induce synthesis of components for synthesizing RSV virions and, in some cases, may cause morphological transformation of the cultures.

- 4359 FIVE-YEAR ANALYSIS OF A STRAIN OF MAMMARY ADENOCARCINOMA: MALIGNANCY, STRUCTURE, VIRAL PARTICLES. (Fr.) Thomas, J. A. (Lab. Cell Biol., U. Paris, France), E. Hollande, M. Henry and C. Jouanne. *C R Acad Sci [D] (Paris)* 277(3):381-386, 1973.

The T III strain of mouse mammary adenocarcinoma has been passaged for 5 yr in an inbred strain (LAB) of Swiss mice. After 92 continuous passages an average of 86.7% of the mice inoculated developed tumors which, in 14.5% of the cases, regressed. Beginning with the 20th passage tumor invasion and metastases were no longer observed, but the histological structure of these tumors has remained unchanged. Mammary tumor virus particles (A and B) were present in large numbers in the initial tumor, but C particles only formed during some stages of budding on the plasma-membrane; very few Ai particles were detected. During the first 28 passages A-particles were always present, but B, C and Ai particles were not detected in some passages. From the 42nd to the 68th passage, C-particles were present in the largest numbers and the number of Ai particles had increased. Starting with the 42nd passage, B particles disappeared, and A particles disappeared between the 77th and the 91st passages; large numbers of C particles were detected, and intracisternal Ai particles were found near C particles after the disappearance of A and B particles. Between the 30th and 89th passages hemorrhagic ascites occurred in 42 of the 316 mice which developed abdominal adenocarcinomas when injected i.p. with the virus. Ascites fluid was reinoculated in ten cases, and two ascites strains of T III were obtained, but only one could be passaged for any length of time. The ascites fluid contained cancer acini. C and Ai particles were present in the acini, but A and B particles had disappeared.

- 4360 THERMOSENSITIVE EVENTS IN ADENOVIRUS TRANSFORMED CELLS. II. HELPING OF ADENOVIRUS-ASSOCIATED VIRUSES. (E.) Lefebvre, N. (Inst. Pasteur Brabant, Brussels, Belgium), M. P. Beumer-Jochmans and S. Sprecher-Goldberger. *Arch Gesamte Virusforsch* 40:248-254, 1973.

Using the indirect immunofluorescence assay, diffuse herpetic antigens were demonstrated in hamster cells infected with the Herpes simplex type 2 (HSV₂) virus at 35 or at 37 C. A similar picture was obtained with adenovirus transformed hamster cells after herpesvirus infection at 35 C, but at 37 C fluorescence was restricted to the perinuclear region. Coinfection of the transformed cells with HSV₂ and adenovirus-associated virus type 3 (AAV₃) produced the appearance of AAV₃ antigens at 35 C but not at 37 C. Coinfection of non-transformed hamster cells produced AAV₃ antigens which were more intense at 37 C than at 35 C. The thermosensitive event which is necessary for helping AAV₃ may be the same as that necessary for the appearance of herpes antigen(s) in the peripheral part of the cell. It is believed that HSV and AAV compete for an HSV-induced product that is required for the replication of both viruses and that the required product is induced in the cell, rather than present in the parental viruses, since the event has different characteristics in two types of cells: it is thermosensitive in the transformed cells while it is even more efficient at 37 C than at 35 C in the non-transformed cells.

- 4361 LIGAND-INDUCED REDISTRIBUTION OF CONCAVALIN A RECEPTORS ON NORMAL, TRYPSINIZED AND TRANSFORMED FIBROBLASTS. (E.) De Petris, S. (Zool. Dept., U. Coll. London, England), M. C. Raff and L. Mallucci. *Nature [New Biol]* 244(139):275-278, 1973.

It was suggested in previous work that the increased concanavalin A (con-A) agglutination of transformed cells compared to normal cells results from differences in the arrangements of cell surface lectin receptors in the different types of cells. Labelling of receptors with con A-ferritin (con A-FT) or with con A-peroxidase had indicated random distribution of receptors in normal cells, clustered distribution in transformed cells. To determine whether the clustered distribution in transformed cells may have been secondarily induced by the binding of the multivalent con A, attempts were made to correlate the pattern of induced redistribution and agglutinability in normal, trypsinized and transformed cells, using thin section electron microscopy of con A-FT-treated cells. The cell systems tested were normal mouse 3T3 cells, 3T3 cells transformed by polyoma virus (PV-3T3), Balb/c secondary mouse embryo fibroblasts (MEF), C57BL/6 tertiary mouse embryo fibroblasts, and C57BL/6 fibroblasts transformed by polyoma virus (PV-TT3). In certain experiments cells prefixed with glutaraldehyde were used. This was expected to immobilize surface proteins without substantially altering the ability of the carbohydrate moiety of glycoproteins to bind con A. Normal 3T3 cells were only slightly agglutinated by con A, while transformed PV-3T3 and, even more

markedly, trypsinized 3T3 cells were strongly agglutinated. Normal MEF were also agglutinated, although agglutination was enhanced after trypsinization. When prefixed cells were labelled with con A-FT, the FT molecules appeared to cover all parts of the membrane, including microvilli, without appreciable differences between normal, trypsinized or transformed cells. When cells were incubated with con A-FT before fixation, the distribution was no longer uniform and ferritin was irregularly concentrated in patches of variable size. Further, the amount of labelling and the degree of patching were not appreciably different in normal, trypsinized or transformed cells, in spite of their marked differences in agglutinability. It was concluded that the patching of con A receptors is a secondary phenomenon, probably induced by cross linking by the multivalent con A. This was further supported by the enhancement of patching by anti-FT antibodies. The results suggest that clustering of receptors, although perhaps important and even necessary for agglutination, is not in itself sufficient to explain differences in agglutination, at least when induced by con A-FT. Morphological characteristics of the cell surface, such as number and distribution of microvilli, could influence agglutination, but cannot explain the difference in agglutination between normal and trypsinized cells where these features were similar.

4362 INCREASE OF EB VIRUS POSITIVE CELLS IN CULTURED BURKITT LYMPHOMA CELLS AFTER SHORT-TIME EXPOSURE TO HEAT. (E.) Vonka, V. (Inst. Sera Vaccines, Prague, Czechoslovakia) and L. Kutinova. *Neoplasia* 20(3):349-352, 1973.

Cultured Burkitt lymphoma cells were examined by fluorescent microscopy for the percentage of Epstein-Barr virus positive cells (EB3) after heat treatment. In cultures exposed to 43 or 45 C for 15 or 45 min, the percentage of immunofluorescence (IF) positive cells (2.7-3.2%) was two to three times higher than in untreated cultures or those exposed to lower or higher temperatures. No IF positive cells were detected among heated or unheated virus-negative lymphoblastoid cells (NC37). Separate treatment with cis-dichlorodiammineplatinum (II) (DDP, 3.2 µM) or heat (45 C for 30 min) had a similar effect on the percentage of IF positive cells (3.2 and 3.1, resp.). In cultures treated with both DDP and heat, the percentage of positive cells was not higher than in those treated separately. The mechanism for the increase of IF positive cells after heat treatment is not understood, but it is possible that heat led to inactivating a repressor of some EBV genome functions necessary for viral antigen synthesis.

363 ISOLATION AND CHARACTERIZATION OF REVERTANT CELL LINES. IV. DIRECT SELECTION OF SERUM-REVERTANT SUBLINES OF SV40-TRANSFORMED 3T3 MOUSE CELLS. (E.) Vogel, A. (Cold Spring Harbor Lab., N.Y.) and R. Pollack. *J Cell Physiol* 82(2):189-198, 1973.

SV101, the sarcoma virus 40-transformed subline of the

mouse fibroblast line 3T3, is both serum- and density-transformed, since it grows in both 1% and 10% calf serum, and grows beyond confluence in 10% calf serum. Negative selection at low cell density in 1% calf serum or in 10% gamma-depleted serum permits direct recovery of serum-revertant sublines of SV101. These sublines are unable to grow in 1% calf serum. Although negative selection at high cell density in 10% calf serum is known to permit recovery of density-revertant sublines of SV101, most density-revertants selected with FUdr or BUdr are not serum-revertant. However, all serum-revertants isolated so far are density-revertant as well. As has been reported for density revertants, serum revertants contain more DNA and more chromosomes/cell than their SV101 parent.

4364 INDUCTION OF ONCORNAVIRUSES IN HUMAN AND ANIMAL CELLS. (E.) Altstein, A. D. (Ivanovsky Inst. Virol., Acad. Med. Sci., Moscow, USSR), L. G. Zakharova, R. M. Argirova, S. F. Gerassina and V. M. Zhdanov. *Neoplasia* 20(5):545-550, 1973.

Oncornavirus-like particles were induced in several cell systems after treatment with 5-iodo- and 5-bromodeoxyuridine (IUdR and BUdR). These particles were found in tissue culture fluids 5-6 days after the treatment of mouse (C57B1/6) and rat (WAG) cell lines with thymidine analogue or after long-term cocultivation of treated and untreated cells (FK and HEL-3 cells), thus indicating a long term reproduction of these particles. Particles containing reverse transcriptase were not induced in two continuous human cell lines (AS and 709) and in two human embryo lung cell strains (HEL-1 and HEL-4). Reproduction of particles containing reverse transcriptase was found however in the cell strain HEL-3 following long-term cocultivation of treated and untreated cells. The results suggest that human cells are characterized by a less inducibility of oncornavirus genetic material by thymidine analogues as compared to rodent cells. A hypothesis is proposed to explain the high inducing activity of halogen-containing thymidine analogues incorporated into DNA instead of thymidine.

4365 EARLY PRODUCTION OF ROUS SARCOMA VIRUS IN COCULTIVATED VIROGENIC MAMMALIAN AND CHICK EMBRYO CELLS. (E.) Obukh, I. B. (Acad. Med. Sci., Moscow, USSR), A. D. Altstein and I. N. Kryukova. *Neoplasia* 20(5):551-553, 1973.

Virogenic mammalian tumor cells were cocultivated with chick fibroblasts after the cell mixture was treated with inactivated Sendai virus to form heterokaryons. The virogenic cells used were XC cells, H-67 cells of a Syrian golden hamster tumor induced by Carr-Zilber strain serially passaged *in vitro* for 5 yr, HRO-analogous culture and No. 36-mouse embryo cell culture (mouse line A) transformed by Carr-Zilber strain. Sendai virus was inactivated by β-propiolactone. The presence of infectious Rous sarcoma virus (RSV) was determined by inoculation of cell extracts into 5 day old white Leghorn chicks. Infectious RSV was found in mixed cultures as early

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as 2-3 hr after cocultivation. No increase in virus amounts was noted during 24 hr observation period. The results obtained indicate that the minimal latent period of RSV maturation following cell fusion is 2-3 hr, that is, 8-10 times shorter than in the usual infectious system "RSV-chick fibroblasts". It is suggested that virogenic mammalian tumor cells contain RSV precursor and it takes the latter 2-3 hr to mature in the heterokaryon.

- 4366 ASSOCIATION BETWEEN MAREK'S HERPESVIRUS AND HUMAN CANCER. I. DETECTION OF CROSS-REACTING ANTIGENS BETWEEN CHICKEN TUMORS AND HUMAN TUMORS. (E.) Makari, J. G. (Makari Res. Lab., Englewood, N.J.). *Oncology* 28(2):164-176, 1973.

Common antigenicity was discovered between glycoprotein antigens obtained from leukotic lesions of chickens with Marek's disease and antigens obtained from a variety of human cancers. Schultz-Dale reactions were positive in 41% of tests, based on 20 tumors, when glycoprotein antigen derived from the GA strain of Marek's disease was used as the immunizing antigen and tumor antigen was used as the releasing antigen. When antigen from the CR64 strain of Marek's disease was the immunizing antigen, 34% of tests, based on 14 tumors, were positive. For the GA strain antigen, cross-reactivity was highest for the leukemia-lymphoma-Hodgkin's disease (49%) and gastrointestinal carcinoma groups (49%). For the CR64 strain antigen, cross-reactivity was highest for the genitourinary (46%) and gastrointestinal carcinoma groups (41%). These results suggest that Marek's disease herpesvirus is a possible etiological agent in human cancer.

- 4367 LATENT PERIOD IN LEUKAEMIA INDUCTION BY THE RADIATION LEUKAEMIA VIRUS. (E.) Haran-Ghera, N. (Dept. Exp. Biol., Weizmann Inst. Sci., Rehovot, Israel). *Nature [New Biol]* 238(79):21-23, 1972.

Radiation leukemia virus was injected i.p. into 15 litters of newborn C57BL/6 mice, and the animals killed 6 to 90 days later. Cell-free extracts were prepared from the thymus, spleen, bone marrow, lungs, and kidneys and their leukemogenic activity evaluated by injecting the extract into adult thymus and exposing the inoculated mice to whole-body irradiation. Extracts prepared from the thymuses of 16-day-old animals induced leukemia in 100% of inoculated adults, while bone marrow and whole blood extracts were less active. Extracts from the lungs, kidneys, spleens, bone marrow, and whole blood of 21-day-old mice showed a 35 to 60% leukemogenic activity, while the thymus maintained its 100% activity. Only thymus extracts from 30-, 60-, and 90-day-old animals remained leukemogenic. In another experiment, immunization of C57BL/6 mice by the radiation leukemia virus, which was injected when the animals were less than 16 hours old, was evaluated by testing host resistance against isotransplantation of 1,000,000 leukemia cells induced by the same virus. The tumor cell takes were much lower

in the virus inoculated hosts than in the controls. However, the phenomenon of host resistance was no longer demonstrable in mice inoculated at 190 days of age (the average latent period for leukemia development with this same virus preparation is 185 days). In the third experiment, the neutralization capacity of the serum collected from C57BL/6 mice 90, 120, 150, or 180 days post-virus inoculation was evaluated by testing its influence on the leukemogenic activity of the radiation leukemia virus. The serum taken from hosts 90 to 150 days after virus inoculation reduced the virus leukemia induction capacity from 75-90% to 0-15%; serum collected from 180-day-old mice had no neutralizing effect on the virus. Thus, for a few months after virus inoculation, newborn mice are immunized, with this condition preventing the proliferation of transformed leukemic cells within this latent period.

- 4368 EFFECT OF CELL CHROMOSOME NUMBER ON SIMIAN VIRUS 40 REPLICATION. (E.) Robb, J. A. (Dept. Path., U. California, San Diego, La Jolla) and K. Huebner. *Exp Cell Res* 81:120-126, 1973.

Simultaneously cloned monkey CV-1 cell sublines were found to differ in morphology, cloning efficiency, chromosome number, and sensitivity of Simian virus 40 (SV40) virion productive infection. A fibroblast-like clone, FC7, when compared with an epithelioid clone, TC7, had a lower mean chromosome number and was resistant to SV40 virion infection. Virion adsorption and penetration were similar in both the FC7 and TC7 cells, and both cell types were equally sensitive to first cycle SV40 DNA infection. As the subdiploid mean chromosome number of the FC7 cells increased with passage time toward the TC7 subtetraploid number, the FC7 cells became more sensitive to virion infection. This host gene-dosage effect on SV40 productive infection suggests that a monkey cell function participates in the SV40 uncoating and/or viral genome activation process.

- 4369 ISOLATION OF AN RD-114-LIKE ONCORNAVIRUS FROM A CAT CELL LINE. (E.) Fischinger, P. J. (Natl. Cancer Inst., Bethesda, Md.), P. T. Peebles, S. Nomura and D. K. Haapala. *J Virol* 11(6):978-985, 1973.

A clone of cells derived from a continuous line of cat cells (CCC) spontaneously produced an RNA C-type virus (CCC virus) which did not have the group-specific antigen of the standard strains of feline leukemia viruses but did have that of the RD-114 virus. Single hit infection of a virus-yielding CCC cell with only the feline leukemia virus pseudotype of murine sarcoma virus [MSV(FeLV)] resulted in the release of a pseudotype of MSV coated with the CCC virus envelope. Host range, transmission of virus, helper functions, interference properties, and specific neutralization showed that the CCC and the RD-114 isolates, as well as their respective MSV pseudotypes, are closely related if not identical. Parental, virus-negative cells frozen before the existence of RD-114 were

induced by 5-iododeoxyuridine to yield CCC-like virus *de novo*. Infection of susceptible human cells with the chemically induced virus resulted in interference with the CCC virus pseudotype of MSV but not with the FeLV pseudotype of MSV.

- 4370 PROTECTIVE ACTION OF POLYINOSINIC-POLYCYTIDYLIC ACID IN VIRAL AND TRANSPLANTED LEUKEMIAS IN MICE. (It.) Chieco-Bianchi, L. (Inst. Path. Anat., U. Padua, Italy), D. Collavo, G. Biasi and A. Colombatti. *Tumori* 59(3):167-180, 1973.

Newborn RFM/Un mice, injected with leukemogenic Graffi virus, received 0.02 mg polyinosinic-polycytidylic acid (Poly I:C) i.p., twice a wk for two months starting 24 hr after virus injection. Leukemia incidence in treated mice was significantly reduced, i.e., 56% with a mean latent period of 19 wk as compared to 87% and 15 wk latency in controls. Adult RFM/Un mice, injected i.v. with different doses of two transplantable syngeneic leukemias, received four doses of 0.1 mg Poly I:C on alternate days starting 24 hr after cell transplant. The evaluation of neoplastic nodules on the spleen surface according to the method of Bruce and Van Der Gaag was used to evaluate leukemic growth. Mice receiving Poly I:C showed a marked reduction in spleen nodule counts for all cell doses of both leukemia lines, and histological examination of the spleen confirmed an actual decrease in leukemic foci and disclosed enlargement of germinal centers and periarteriolar areas of lymphoid follicles. To investigate whether Poly I:C is capable of abolishing the state of immunological tolerance to cellular virus-induced antigens, adult (CBA x C57BL)F₁ mice, injected neonatally with Graffi virus, received 0.20 mg Poly I:C i.p. together with 10⁴ or 10⁵ cells from a syngeneic transplantable leukemia originally induced by Graffi virus. No differences in leukemic deaths were observed between Poly I:C treated virus-injected animals and controls, even though normal mice receiving Poly I:C and 10⁴ cells showed 50% survival compared to 0% in control groups. Leukemic cells incubated *in vitro* in Hank's Eagle Minimum Essential Medium containing 0.1, 1, 10 or 100 µg Poly I:C/ml for 2, 4, 8 and 24 hr (37 °C, 5% CO₂) did not exhibit significant changes in viability as evaluated by phase contrast microscopy. It is concluded that while interferon production by synthetic RNA may be a major inhibiting factor in virus-induced tumors, nonspecific stimulation of immunity and direct effects on cell metabolism and replication may play a significant role in other neoplastic conditions.

- 4371 ISOLATION AND CHARACTERIZATION OF REVERTANT CELL LINES. III. ISOLATION OF DENSITY-REVERTANTS OF SV40-TRANSFORMED 3T3 CELLS USING COLCHICINE. (E.) Vogel, A. (Cold Spring Harbor Lab., N.Y.), R. Risser and R. Pollack. *J Cell Physiol* 82(2):181-188, 1973.

Treatment of the sarcoma virus 40 (SV40) transformed 3T3 cell line, SV101, with 0.5 µg/ml colchicine permits the isolation of polyploid revertant sublines which have lower saturation densities in 10% calf serum than

SV101. These low saturation density lines have also reverted to a high serum requirement for growth, and are unable to form colonies in methocel. Normal SV40 has been recovered from these revertants. All colchicine revertants contain more chromosomes than SV101, with modal values varying from 80-90 chromosomes/cell. The DNA content/cell of the revertants is also increased. 3T3 cells are more resistant to colchicine than SV3T3 cells at all cell densities. Colchicine revertants do not display a 3T3-like resistance to colchicine at low density, but do survive colchicine at confluent cell densities, presumably due to their increased contact inhibition.

- 4372 MALIGNANT TRANSFORMATION OF BHK21 CLONE 13 CELLS BY BK VIRUS--A HUMAN PAPOVAVIRUS. (E.) Major, E. O. (U. Illinois Med. Ctr., Chicago) and G. Di Mayorca. *Proc Natl Acad Sci* 70(11):3210-3212, 1973.

Colony growth in soft agar was used to transform BHK21 clone 13 cells by BK virus, a widespread human papovavirus. This malignant transformation of BHK21 clone cells by BK virus *in vitro* was compared with the transformation of BHK21 clone 13 cells by a known oncogenic papovavirus, polyoma. With both viruses, transformation appeared to be linearly dependent upon the dose of virus, although when multiplicities greater than 100 to 300 plaque-forming units per cell were used, the transformation frequency reached a plateau; in this characteristic, the human papovavirus resembles polyoma virus. However, there appeared to exist a substantial difference between BK and polyoma viruses in the ratio of small, pin-point colonies to larger colonies which appear in soft agar; the BK virus produced more small colonies than large colonies, while the reverse was true of the polyoma virus. The small-colony variant probably represents abortive viral transformants, while the large-colony variants appear to represent truly malignant transformed cells. It is concluded that BK is an oncogenic virus.

- 4373 MEMBRANE PROTEINS OF UNINFECTED AND ROUS SARCOMA VIRUS-TRANSFORMED AVIAN CELLS. (E.) Bussell, R. H. (Dept. Med., Stanford U., Calif.) and W. S. Robinson. *J Virol* 12(2):320-327, 1973.

Large membrane fragments rich in cell ghosts from uninfected and Rous sarcoma virus (RSV)-transformed chicken embryo fibroblast (CEF) cells were isolated and the membrane proteins separated by electrophoresis in sodium dodecyl sulfate (SDS)-containing gels. A change in the membrane proteins of uninfected cells was regularly observed after transformation with RSV (Rous-associated virus (RAV-1)). A major radioactive protein component of uninfected cells, with a molecular weight of about 142,000, was absent or greatly reduced in amount in membrane preparations from RSV (RAV-1)-transformed cells. The radioactive membrane component, MP-1, was present in the same amount relative to other membrane proteins in populations of rapidly grow-

ing CEFs and in confluent monolayers in which most cells are not dividing, which suggests that cell growth rate does not differentially affect the synthesis of this component. Infection of cells with a transforming virus, RSV(RAV-1), but not with a nontransforming virus such as RAV-1, was associated with the reduction or disappearance of this component. This suggests that the viral transformation of cells, rather than avian leukosis virus replication in cells, is necessary for the observed alteration in MP-1. Experiments with radioactive fucose and glucosamine suggest that MP-1 is a glycoprotein.

- 4374 PRIMATE RNA TUMOR VIRUS-LIKE DNA SYNTHESIZED ENDOGENOUSLY BY RNA-DEPENDENT DNA POLYMERASE IN VIRUS-LIKE PARTICLES FROM FRESH HUMAN ACUTE LEUKEMIC BLOOD CELLS. (E.) Gallo, R. C. (Nat'l. Cancer Inst., Bethesda, Md.), N. R. Miller, W. C. Saxinger and D. Gillespie. *Proc Nat Acad Sci* 70(11):3219-3224, 1973.

Subcellular cytoplasmic particles from fresh human leukemic myeloblasts were assayed for absorbance at 260 nm and for endogenous RNA-dependent DNA polymerase activity. Some of the particles were then used to generate (3H)DNA, while others were purified by repeated isopycnic banding and the enzymatically active fractions used to synthesize endogenous (3H)DNA. This procedure produced a particle of discrete biochemical composition having a density of 1.16 to 1.17. This particle endogenously synthesized DNA via an RNA primer and template. About half of the DNA sequences synthesized in the presence of actinomycin D hybridized to the RNA isolated from the type C sarcoma viruses of primates and mice: lower annealing values were obtained with RNA isolated from other sarcoma or leukemia viruses. These data support the contention that if RNA tumor viruses are involved with human cancer, they are likely to be immature or replication-defective in the sense that they can only be recovered as intracellular entities.

- 4375 STRUCTURAL CHARACTERISTICS AND NUCLEOTIDE SEQUENCE ANALYSIS OF GENOMIC RNA FROM RD-114 VIRUS AND FELINE RNA TUMOR VIRUSES. (E.) East, J. L. (U. Texas System Cancer Ctr., M.D. Anderson Hosp. Tumor Inst., Houston), J. E. Kneseck, P. T. Allen and L. Dmochowski. *J Virol* 12(5):1085-1091, 1973.

To determine whether RD-114, a type C virus, might be a feline virus, the structural characteristics of genomic RNA from RD-114 were compared with those of genomic RNA from known feline RNA tumor viruses (sarcoma, leukemia, and Crandell virus), and the extent of nucleotide sequence homology between the RNA genome of RD-114 and the RNA genomes of feline RNA tumor viruses was measured by nucleic acid hybridization. The RNA genomes of RD-114 and Crandell virus were found to have similar sedimentation characteristics and share common nucleotide sequences. The RNA of released RD-114 virions harvested at either 4 or 24 hr had a

sedimentation coefficient of 50S, which was identical to that of the genomic RNA of Crandell virus. The absence of a structural rearrangement of the RNA genome within released RD-114 virions appeared to be a unique structural characteristic that was not shared with the RNA genomes of FeSV-R (feline sarcoma virus) or FeLV-R (Feline leukemia virus). The RNA genome of RD-114 had extensive nucleotide sequence homology with the RNA genome of Crandell virus, but only limited nucleotide sequence homology with the RNA genomes of FeLV-R and FeSV-R. The genetic similarity between RD-114 and Crandell virus indicates that: these two viruses could represent two different strains of endogenous feline RNA tumor viruses that have common nucleotide sequences along their RNA genomes; or RD-114 could be a recombinant virus containing human viral nucleotide sequences in addition to those shared with the RNA genome of Crandell virus.

- 4376 SUBVIRAL COMPONENTS OF A WILD MOUSE EMBRYO-DERIVED TYPE C ONCORNAVIRUS. (E.) Pal, B. K. (U. Southern California Sch. Med., Los Angeles), M. Wright, J. E. Officer, M. B. Gardner and P. Roy-Burman. *Virology* 56(1):189-197, 1973.

A wild mouse embryo-derived nontransforming type C virus, WM-1504E, which showed *in vivo* neurotropic and lymphomagenic activities, was analyzed for biochemical and antigenic characterizations. The virions contained two major species of RNA, 70 S and 4 S. The 70 S RNA was heat dissociated into a heterogeneous species with a peak distributions at 35 S, 18 S, and 4 S regions. The gel filtration pattern in guanidine-hydrochloride and neutralization by antibody against murine leukemia virus (MuLV) polymerase of the virion associated reverse transcriptase resembled those of MuLV enzyme. Virions contained six major protein groups as detected by agarose gel filtration in guanidine-hydrochloride. The two largest proteins were glycoproteins, and the next largest protein contained the group-specific antigenic determinants of murine type C oncornaviruses. The approximate molecular wt of the major proteins as determined by SDS-polyacrylamide gel electrophoresis were: 100,000 (and 11,000), 76,000, 29,000, 16,000, 13,500, and 11,000 daltons. The last group also contained a polypeptide of 10,000 daltons. These values correspond closely to those of MuLV (Moloney) proteins. It is concluded that WM-1504E virus represents a murine type C oncornavirus.

- 4377 PROPERTIES OF MOUSE EMBRYO CELLS INFECTED WITH MURINE SARCOMA VIRUS AND SIMIAN VIRUS 40 SIMULTANEOUSLY *IN VITRO*. (E.) Simons, P. J. (Tobacco Res. Council Labs., Harrogate, Yorkshire, England). *J Gen Virol* 19(3):411-416, 1973.

Some properties of cells derived from Balb/c amitotic cell cultures infected simultaneously with simian virus 40 (SV40) and murine sarcoma virus-Harvey (MSV-H) were investigated. Both the SV40-infected cells (SV cells) and the SV40-MSV-H-infected cells (SMT cells) grew rapidly in monolayer culture. SV cells were epithelioid in appearance, with the nuclei

containing an increased number of nucleoli. Cultures of passaged SMT cells consisted of small densely staining, spindle cells, with occasional larger epithelioid cells. SMT cells liberated large amounts of MSV-H into the culture medium. Neither the SV nor SMT cells released infectious SV40 into the culture fluids. Virtually all nuclei (98%) of 8th passage SMT contained SV40 T antigen, whereas only 30% of 8th passage SV cell nuclei contained T antigen. When administered s.c. to weanling mice, SMT and MSV-H-infected (MT cells) released infectious MSV. In some animals the injected cells grew rapidly and a proportion of these animals died of splenic rupture after a period of more than 50 days. Almost all of the animals inoculated with MSV-producing cells, but not developing tumors at the site of inoculation, eventually died of splenic rupture and hemorrhage. None of the animals receiving the SV40 cells developed tumors or died over a 6-month period. The results show that two tumor viruses of different biological type, each of which can transform fibroblastic cells in culture, act synergistically in the same cell. The evidence suggests that the SMT cells may have been synthesizing MSV at a faster rate than cells infected with MSV alone.

4378 ENHANCEMENT OF FRIEND VIRUS LEUKEMIA BY LEUCOGENENOL. (E.) Elliott, S. C. (Biol. Dept., Drake U., Des Moines, Iowa), A. N. Jacoby, M. A. Barnhill and W. E. Howard. *J Natl Cancer Inst* 51(4):1135-1139, 1973.

The effects of leucogenenol ($C_{18}H_{25}NO_8$) on Friend virus (FV) leukemia were studied in female BALB/c mice. Leucogenenol given before, before and after, or after FV infection augmented the leukemic process. That leucogenenol potentiated the leukemia in test animals was shown by higher peripheral nucleated cell counts, higher spleen and liver weights, more abnormal cells in peripheral differentials and, in one set of animals, rapid mortality. The elevated humoral antibody titer in FV-infected animals given leucogenenol is ascribed an important role in potentiating the disease. It is also suggested that leucogenenol increased a stem-cell pool and thus enhanced both B-cell immunogenesis and leukemogenesis.

4379 INDUCTION OF TYPE C VIRUS-RELATED FUNCTIONS IN NORMAL RAT EMBRYO FIBROBLASTS BY TREATMENT WITH 5-IODODEOXYURIDINE. (E.) Verwoerd, D. W. (Natl. Cancer Inst., Bethesda, Md.) and P. S. Sarma. *Int J Cancer* 12(3):551-562, 1973.

Secondary cultures of rat embryo fibroblasts derived from six different strains of normal laboratory and wild rats (Osborne-Mendel, Fisher, ACI, Lewis, Wistar-Furth, and wild), were treated with 5-iododeoxyuridine in an attempt to establish the presence of endogenous type C viruses in rat cells. All the cell lines tested responded with a transient appearance of RNA-dependent DNA polymerase (RDP) activity which reached a peak 3 days after the beginning of treatment. However, no viral

particles or rat gs-antigen could be detected at this stage. In the cells derived from two highly inbred strains (ACI and Wistar-Furth), a spontaneous second burst of RDP activity was observed after 10-12 days, and, on subculturing these cells, a 20- to 40-fold increase of enzyme activity was obtained. During this peak of activity, both gs-antigen and viral particles capable of incorporating labelled uridine were detected. Electron microscopic examination revealed the presence of complete or immature virions in one cell strain. The virus induced was non-infective for monolayer cultures of ten different animal species. Attempts to rescue infective virus by complementation with murine leukemia or sarcoma viruses failed. Prolonged cultivation of the treated cultures did yield cells with transformed morphology, but no tumors were induced by inoculation of these cells into newborn rats of various inbred strains, with and without immunosuppressive treatment.

4380 DIFFERENTIAL EXPRESSION OF TRANSFORMATION IN RAT AND CHICKEN CELLS INFECTED WITH AN AVIAN SARCOMA VIRUS *ts* MUTANT. (E.) Graf, T. (Duke U. Med. Ctr., Durham, N. C.) and R. R. Friis. *Virology* 56(1):369-374, 1973.

Cells of the rat NRK line were infected with *ts* 339, a mutant of avian sarcoma virus B77, and clones were isolated which displayed the characteristics of transformed cells at 33 C. These cells reverted to a normal phenotype at 37 C. In comparison, chicken cells infected with the same mutant remained transformed at 37 C and acquired properties of normal cells only after temperature shift to 41 C. Several possibilities may serve as an explanation for the phenomenon observed: 1) the activity of the heat sensitive, virus specific molecule could be affected by factors in the microenvironment such as pH or the concentrations of certain ions, which could differ in avian and mammalian cells; 2) there could be a difference in receptor sites for the heat sensitive molecule required for transformation; and 3) since avian sarcoma viruses merely establish an abortive infection in mammalian cells, it appears possible that they induce less transforming molecules in mammalian than in chicken cells. The "leakiness" of the temperature sensitive molecule might thus be greater in the chicken system and could be reflected in an increased heat resistance of the transformation process in these cells.

4381 PROPERTIES OF AN ONCORNAVIRUS GLYCOPROTEIN: EVIDENCE FOR ITS PRESENCE ON THE SURFACE OF VIRIONS AND INFECTED CELLS. (E.) Kennel, S. J. (Scripps Clin. Res. Fdn., La Jolla, Calif.), B. C. Del Villano, R. L. Levy and R. A. Lerner. *Virology* 55(2):464-475, 1973.

The surface proteins of a lymphoblast line (SCRF 60_A) from an NZB mouse were studied. Iodination of the cell surface followed by solubilization and immunoprecipitation with antisera prepared against various murine leukemia viruses and cells produced a precipitate which gave a single peak in SDS-polyacrylamide gel electrophoresis and comprised about 5% of the incorporated iodide. The antigen is a glycoprotein

of apparent molecular wt (MW app) of approximately 70,000 daltons and comprises about 0.1% of the total cellular amino acids and about 10% of the cellular glucosamine. Studies on the oncogenic C type virus produced by SCRF 60_A, Scripps leukemia virus (SLV), showed that a glycoprotein of identical radioactive labeling properties and SDS-polyacrylamide gel electrophoresis mobility is present on the surface of the virions. It constitutes about 10% of the virion amino acids and about 50% of the glucosamine. This protein reacts with sera which neutralize Moloney, Kirsten, Rauscher, AKR, and, of course, Scripps viruses. These data suggest that this is the antigen detected when murine leukemia cells are studied by immunofluorescence and that this antigen may be involved in virus neutralization.

- 4382 ISOLATION AND PRELIMINARY CHARACTERIZATION OF THE RNA-CONTAINING R-TYPE, VIRUS-LIKE PARTICLE OF BHK-21 CELLS. (E.) Abu, E. (Georgetown U. Sch. Med. and Dentistry, Washington, DC) and K. V. Holmes. *J Virol* 12(5):1164-1172, 1973.

Ultracentrifugation and polyethylene glycol precipitation were used to isolate an R-type virus-like particle (VLP) from the medium of BHK-21-F cells derived from baby hamster kidney: RNA was then extracted from the VLP. The VLP contained labile, single-stranded RNA with a molecular weight of about 10^7 and a sedimentation constant of 60 to 70S in sucrose density gradients. The R-type VLP could be labeled with 3H-uridine but not with 14C-thymidine and 3H-uridine was incorporated into the VLP in the presence of actinomycin D, indicating that the VLP is an RNA-containing particle which replicates from an RNA template. Although the virions of the R-type VLP are similar in size to those of AMV, a member of the oncornavirus group, their appearances in thin sections are quite different, as are their sites of replication and their densities: thus the R-type VLP should not be classified with the oncornavirus group. Preliminary studies indicate that the R-type VLP also differs significantly from the corona virus group.

- 4383 STRANDEDNESS AND COMPLEMENTARITY OF DNA FROM LONG-TERM RNA-DEPENDENT DNA POLYMERASE REACTIONS OF SOEHNER-DMOCHOWSKI MURINE SARCOMA VIRUS. (E.) East, J. L. (M.D. Anderson Hosp. Tumor Inst., Houston, Texas), J. E. Knesek, P. T. Allen and L. Dmochowski. *J Virol* 12(5):1049-1064, 1973.

The DNA product of the endogenously instructed RNA-dependent DNA polymerase reaction of murine sarcoma virus continued to be synthesized for as long as 64 hr in the presence of 0.008% Triton X-100. Higher detergent concentrations and actinomycin D inhibited DNA product synthesis. The DNA product from long-term polymerase reactions consisted of small DNA fragments as shown by sedimentation in alkaline sucrose gradients. The enzymatic DNA product was separated into a slow sedimenting fraction and a fast sedimenting fraction by rate-zonal centrifugation. Fast sedimenting DNA was the predominant fraction

made in viral polymerase reactions containing 262 mM NaCl. By using a combination of S-1 nuclease and pancreatic RNase A, the amount of single-stranded DNA, double-stranded DNA, and DNA-RNA hybrid present in the slow-sedimenting and fast-sedimenting fractions was determined. Under standard polymerase conditions of 70 mM NaCl, single-stranded DNA was the major form of DNA found in both fractions. In contrast, the prevalent form of DNA made in the presence of 262 mM NaCl was DNA-RNA hybrid. Hybridization studies in which either S-1 nuclease or pancreatic RNase A was used to measure hybrid formation demonstrated not only that the DNA product was complementary in base sequence to the RNA genome, but also that at least 79 to 84% of the RNA genome was transcribed into complementary DNA.

- 4384 IN VITRO PRODUCTION OF MOUSE MAMMARY TUMOR VIRUS IN A MOUSE MAMMARY TUMOR ASCITES LINE. (E.) Keydar, J. (Dept. Microbiol., U. Tel-Aviv, Israel), Z. Gilead, J. R. Hartman and Y. Ben-Shaul. *Proc Natl Acad Sci USA* 70(10):2983-2987, 1973.

An ascites line derived from a spontaneous mouse mammary carcinoma produces, on explantation and long-term cultivation *in vitro*, large amounts of oncornavirus particles. The yield of virus during the first and second day after explantation is low but increases to high levels after three to four days in culture. The yield of twice-purified virus was 1-2 mg/liter of medium. The virus seemed to be relatively free of C-type particles. The biochemical, biophysical, and electron microscopic characteristics of the virions are described. Molecular hybridization and immunological methods identify these virions as mouse mammary tumor virus.

- 4385 ISOLATION OF TYPES A AND B ONCORNAVIRUSES IN TRANSPLANTABLE CELL LINES FROM HUMAN CERVICAL CARCINOMA. (Rus.) Bykovskii, A. F. (N. F. Gamaleia Inst. Epidemiol. Microbiol., Moscow, USSR), N. V. Klitsunova, G. G. Miller, F. I. Ershov and V. M. Zhdanov. *Vopr Onkol* 19(8):68-72, 1973.

Oncornaviruses were isolated from two clones of HeLa cells (Bristol strain) by ultracentrifugation in density gradients of 20-60% sucrose. Virus was isolated from one clone only after treatment with mitomycin C. Labeling of cultures with 3H-uridine showed most virus was present in the fraction with a density of 1.16 g/ml, but radioactivity in this fraction disappeared when actinomycin D (0.5 µg/ml) was added to the culture medium. Type A and B virions were both formed inside of the cells and on the cell surface, and their outer membranes were made up of elements of the cytoplasmic membrane. In immunodiffusion experiments, immune sera for type B oncornavirus obtained from HEp-2 cells reacted with oncornavirus obtained from one clone of HeLa cells and with that obtained from HEp-2 cells. Further studies are needed to determine whether viruses isolated from HeLa and HEp-2 cells are strains of the same virus or different variants.

- 4386 WIDE HOST RANGE OF MURINE SARCOMA VIRUS.
(E.) Rhim, J. S. (Microbiological Associates, Inc., Bethesda, Md.), M. L. Vernon, F. G. Duh and R. J. Huebner. *Int J Cancer* 12(3):734-741, 1973.

Cells cultures of canine, rabbit, porcine, feline, simian, and human origin were infected with Kirsten murine sarcoma virus (Ki-MSV), an MSV isolate from a rat-passaged murine erythroblastosis virus. Approximately 2 to 3 weeks after infection, morphological alterations were noted in the cultures of rabbit kidney, canine embryo, porcine kidney, feline embryo, and bovine embryo; the cellular morphology remained unchanged in the infected monkey kidney cells and the uninfected controls. The transformed Ki-MSV-infected cells were found to be virus producers; cell-free preparations of supernatant fluid from the *in vitro* altered cultures were infectious to homologous and NRK cells. In addition, the transformed cells contained high titers of group-specific antigen characteristic of the viruses of murine leukemia-sarcoma complex, RNA-dependent DNA polymerase activity was demonstrated in the infected lines, and the *in vitro* cultures contained type-C virus particles. These data indicate that members of the MSV complex, particularly Ki-MSV, exhibit a wider host range than was hitherto believed.

- 4387 TITRATION OF HERPES SIMPLEX VIRUS IN CELL CULTURES DERIVED FROM THE NORMAL CERVIX AND FROM CERVICAL CARCINOMA. (It.) Menarini, L. (Inst. Microbiol., U. Ferrara, Italy), M. Terni, C. Pavanelli and M. Tortora. *Tumori* 59(1):25-32, 1973.

Previous serological investigations have shown a positive correlation between cervical carcinoma and previous infections of the female genital tract with *Herpes simplex* virus (HSV) type 2, but not with type 1. This association could be due to the carcinogenic or cocarcinogenic effect of HSV or to preferential growth of the virus in tumor or transformed cells. To test the latter hypothesis, cells from normal cervix and from cervical carcinoma were cultured *in vitro* and were assayed for HSV-2, HSV-1 and the MP mutant of HSV-1, as were cultures of HEp-2 cells. About the same viral yields and numbers of viral plaques were obtained, and similar plaque morphology and intracellular cytopathic effects were observed in cultures of cells from normal cervix and cervical cancer and in HEp-2 cells. Thus, there is no evidence of preferential viral growth in tumor cells.

- 4388 ALTERATION OF CELL-SURFACE PROTEINS BY VIRAL TRANSFORMATION AND BY PROTEOLYSIS.
(E.) Hynes, R. O. (Imperial Cancer Res. Fund, London, England). *Proc Natl Acad Sci* 70(11):3170-3174, 1973.

The putative cell-surface proteins of the following tissue-culture cells were identified by lacto-

peroxidase-catalyzed iodination, a technique which attaches label only to proteins outside the cell membrane: two clones of the hamster fibroblast cell line NIL.2E (NIL.1 and NIL.8), various clonal derivatives transformed by hamster sarcoma virus (HSV) or polyoma virus (Py), and LX cells. A major exterior cell protein of high molecular weight (band 1) on normal fibroblasts was not detected or detected only in small quantities on the virus-transformed derivatives: similar results were found with chicken-embryo cells transformed by Rous sarcoma virus. This phenomenon was not a clonal variation unrelated to transformation. It is possible that the transformed cells synthesize band-1 polypeptide in reduced quantities or not at all, or they may synthesize it at normal rates but, because it is masked or continually removed from the cell surface, it is not available for iodination. The fact that transformed cells produce proteases coupled with the observation that mild trypsin treatment removes band 1 from normal cells indicates that the failure to iodinate band-1 polypeptide on the transformed cells may be due to its removal by proteolytic digestion. It is possible that transformation leads to the production of proteolytic enzymes which, in turn, allow the transformed cells to escape normal growth controls. In this event, treatment of the transformed cells with proteolytic enzyme inhibitors would tend to render their surface characteristics and growth patterns normal.

- 4389 REVERTANTS OF MOUSE CELLS TRANSFORMED BY MURINE SARCOMA VIRUS. II. FLAT VARIANTS INDUCED BY FLUORODEOXYURIDINE AND COLCEMID. (E.) Nomura, S. (Natl. Cancer Inst., Bethesda, Md.), P. J. Fischinger, C. F. T. Mattern, B. I. Gerwin and K. J. Dunn. *Virology* 56(1):152-163, 1973.

Mouse sarcoma virus-transformed 3T3FL cells (S+L-cells) produced spontaneously and at variable rates flat variants with some properties of nontransformed cells. The frequency of occurrence of such variant cells increased after treatment of S+L- cells with fluorodeoxyuridine (FdUrd) or Colcemid. In one S+L- subline (3-360), the spontaneous flat variants (S) occurred at the rate of about 1 in 80 clonal colonies, and, after treatment with Colcemid or FdUrd, flat variants were observed in 1 of 20 and 30 colonies, respectively. In another S+L- subline (3-321), S variants normally occurred less than 1 in 1000 colonies, and the same treatments increased the frequency to approximately 1 in 70 and 55 colonies, respectively. Both the FdUrd and Colcemid-induced flat variants (F and C) resembled 3T3FL cells morphologically, grew to low saturation densities, and exhibited cloning efficiencies in soft agar which were 10^{-1} to 10^{-4} times lower than that of S+L- cells. However, except for one S subline, they contained murine leukemia group-specific antigen(s) without demonstrable virus production and reverse transcriptase activity. Murine sarcoma virus (MSV) was no longer rescuable by superinfection with murine leukemia virus (MuLV) or by cell fusion with 3T3FL, BALB/3T3, or normal rat kidney cells. All flat variant cells were susceptible to MSV and MuLV infection, and some

degree of enhancement of sensitivity to MSV and MuLV infection was observed in most variant cultures. All flat variant sublines possessed the property of agglutinability by concanavalin A as high as that of S+L- cells. Some flat variant clones spontaneously underwent retransformation during extended cultivation. In contrast to previously described S+L- cell revertants, which spontaneously retransformed morphologically without a rescuable MSV genome, in the present experiments retransformation with a rescuable MSV genome was observed in one of four S sublines and two of five F sublines, but in none of four C sublines. Furthermore, one of five F sublines gave rise to a clone which was flat but which otherwise resembled S+L- cells. In the S and C variants, the loss of expression of transformation was associated with an increase in chromosome number. However, the chromosome number of F variants was similar to or slightly less than that of parental S+L- cells. These studies demonstrated that the reversion of S+L- cells to flat variants occurred by different mechanisms apparently involving either the viral or cellular genes or both, and that some flat variant sublines had retained at least one complete MSV genome in nondetectable form.

- 4390 PREVALENCE OF TYPE-C PARTICLES IN VISCERAL TISSUES OF EMBRYONIC AND NEWBORN MICE. (E.) Vernon, M. L. (Microbiological Associates, Inc., Bethesda, Md.), W. T. Lane and R. J. Huebner. *J Natl Cancer Inst* 51(4):1171-1175, 1973.

Immature budding type-C particles were demonstrated in 59% of the visceral tissues from 72 of 85 embryonic and newborn mice of all strains examined; thus 85% of the embryonic or newborn mice tested had one or more positive tissues. The hematopoietic liver, which develops much earlier than the spleen or thymus, regularly showed relatively large numbers of budding type-C particles as early as 10-14 days gestation. As the spleen and thymus developed, they contributed significantly to the total C-particles count of most strains. Muscle tissue was uniformly negative. The data offer supportive evidence for the transmission of subinfectious virus information from parent to offspring as part of the genetic makeup of normal cells as proposed in the viral oncogene hypothesis. This hypothesis was based in part on the demonstration of expressions of the major polypeptide (group-specific-1) of the type-C RNA tumor viruses in visceral tissue of mouse embryos.

- 4391 LINEAR ASSOCIATION BETWEEN CELLULAR DNA AND EPSTEIN-BARR VIRUS DNA IN A HUMAN LYMPHOBLASTOID CELL LINE. (E.) Adams, A. (Depts. Tumor Biol. Chem., Karolinska Inst., Stockholm, Sweden), T. Lindahl and G. Klein. *Proc Natl Acad Sci USA* 70(10):2888-2892, 1973.

High-molecular-wt DNA from cell line Raji (derived from Burkitt's lymphoma), which contains 50-60 copies of Epstein-Barr virus DNA per cell, was fractionated in neutral solution by several cycles of CsCl gradient centrifugation in fixed-angle rotors. Under the fractionation conditions used,

intact Epstein-Barr virus DNA from virus particles can be separated from the less-dense cellular DNA. In contrast, a large proportion of the intrinsic Epstein-Barr virus DNA component of Raji cells remains associated with cellular DNA, as determined by nucleic acid hybridization. This interaction, which is resistant to Pronase and phenol treatment, is not the result of aggregation. When the molecular weight of Raji DNA is reduced by hydrodynamic shear, the amount of virus DNA associated with cell DNA decreases. However, some virus DNA still remains bound to fragments of cellular DNA after shearing. The association is completely destroyed in alkaline solution. Molecular wt analysis of Raji DNA after denaturation showed that the alkali-induced release of Epstein-Barr virus DNA was specific and not the result of random single-strand breaks. These data indicate that Epstein-Barr virus DNA is linearly integrated into Raji cell DNA by alkali-labile bonds.

- 4392 POLYAMINES AND TUMOR CELLS: EFFECT OF TRANSFORMATION OF CHICK EMBRYO FIBROBLASTS BY ROUS SARCOMA VIRUS ON POLYAMINE LEVELS. (E.) Bachrach, U. (Hadassah Med. Sch., Hebrew U., Jerusalem, Israel), S. Don and H. Wiener. *Biochem Biophys Res Commun* 55(3):1035-1041, 1973.

Chick embryo fibroblast cultures were incubated with B77 or Schmidt Rupin (SR) strains of Rous sarcoma virus (RSV). RSV transformation had a slightly depressive effect on the intracellular levels of spermidine and spermine, while it induced a 3 to 5-fold increase in the cellular putrescine content. Further experimentation showed that the intracellular putrescine remained elevated in the RSV-transformed cells even upon subculturing. Thus, the accumulation of polyamines appears to be an intrinsic property of the malignant cells.

- 4393 CHANGES IN MEMBRANE FUNCTION AND CHROMATIN TEMPLATE ACTIVITY IN DIPLOID AND TRANSFORMED CELLS IN CULTURE. (E.) Baserga, R. (Temple U. Sch. Med., Philadelphia, Pa.), M. Costlow, and G. Rovera. *Fed Proc* 32(11):2115-2118, 1973.

When WI-38 human diploid fibroblasts are stimulated to proliferate by appropriate nutritional changes, the template activity of the chromatin increases within 1 hr after stimulation. After 3 hr, there is an increase in the uptake of cycloleucine, while DNA synthesis begins 12 hours after the initial stimulation. The template activity of the chromatin in SV40 transformed WI-38 (2RA) and spontaneously transformed mouse fibroblasts (3T6) does not increase when these cells are stimulated to proliferate by appropriate nutritional changes. The uptake of cycloleucine increases within 1 hr after stimulation and the length of the pre-replicative phase is 3 hr shorter than in the WI-38 fibroblasts. These and previous data indicate that the WI-38 diploid fibroblasts are in a stationary phase that can be distinguished from the stationary phase of transformed cells like 3T6 and 2RA. It is proposed that the WI-38 cells are in G₀, while the 3T6 and 2RA cells are in G₁, and that the two phases

can be distinguished from each other on the basis of reproducible biochemical differences. It is further hypothesized that transformed and neoplastic cells are less capable of entering the G₀ resting phase than normal diploid cells.

- 4394 ABORTIVE HERPES SIMPLEX VIRUS REPLICATION IN ROUS SARCOMA VIRUS TRANSFORMED CELLS. (E.) Docherty, J. J. (Dept. Microbiol., Pennsylvania State U., University Park), W. R. Mitchell, and C. J. Thompson. *Proc Soc Exp Biol Med* 144(2): 697-704, 1973.

To determine whether a cell transformed by an RNA virus would resist herpes simplex virus (HSV) infection, XC cells evolved from Wistar rat cells were infected with HSV type 1 (HSV-1) and HSV type 2 (HSV-2). Neither virus was able to replicate or significantly induce any virus specific alterations of XC cells. Although both viruses were able to replicate to high levels in non-transformed Wistar rat embryo (WRE) cells and attach to XC cells, they were unable to induce viral proteins, DNA, or TdR kinase, or depress cell DNA synthesis in the XC cell line. The data suggest that prior to viral DNA synthesis, a step in the replicative cycle of HSV was blocked.

- 4395 HERPES-VIRUS AND DOUBLE-STRANDED RNA. (E.) Perez-Bercoff, R. (Inst. Virol., U. Rome, Italy), G. Carrara, A. Dolei and G. Rita. *Experientia* 29(9):1171-1174, 1973.

The mechanism of replication of herpes simplex virus (HSV) DNA was studied. ³H-Uridine labeled viral RNA isolated from infected chick embryo cell (CEC) cultures and purified by LiCl precipitation and gel filtration contained no RNase resistant material, which is characteristic of a double-stranded RNA intermediate. RNA from HSV-infected cells failed to induce interferon production following i.p. injection into adult Swiss mice or following incubation with DEAE-dextran pretreated CEC, mKS-B, MA-104, or HEp-2 cultures, all of which are capable of producing interferon. Pretreatment of cell cultures with HSV-directed RNAs also fails to decrease the extent of replication of vesicular stomatitis virus compared with the extent of replication in infected but untreated control cultures. These results suggest that double-stranded RNA is not present in HSV infected cells and that replication of HSV DNA probably does not proceed via a double-stranded RNA intermediate.

- 4396 PARAMETERS OF INFECTION IN CHICKS EXPOSED TO MAREK'S DISEASE BY TWO DIFFERENT METHODS. (E.) Coles, B. (Dept. Vet. Sci., Washington State U., Pullman), B. R. Cho and S. G. Kenzy. *Poult Sci* 52(5):1918-1923, 1973.

The early parameters of infection were studied in Marek's disease virus (MDV)-infected chickens inoculated intratracheally or exposed for 48 hr to other

disease birds. Viremia and histologic lesions appeared in contact-exposed birds eight days post-exposure compared with 20 days postexposure in the inoculated group. Early lesions were found primarily in the nerves of contact-exposed chicks and in the nerves and skin of inoculated birds. Anti-MDV antibody detectable by the agar gel precipitin and indirect fluorescent antibody techniques was present in the blood of both groups of chicks 24 days after exposure. Birds from each group autopsied at nine wk postexposure did not show a significant difference in the incidence or severity of lesions or in antibody titer. There was no correlation between antibody titer and the incidence of gross lesions.

- 4397 SYNTHESIS OF VIRUS-CAPSID ANTIGEN (VCA) ENHANCED BY ULTRAVIOLET IRRADIATION OF A LYMPHOBLASTOID CELL LINE CARRYING EPSTEIN-BARR VIRUS. (E.) Lai, P. K. (Virus Lab., State Hlth. Lab. Service, Perth, Australia), E. M. Mackay-Scollay and M. P. Alpers. *J Gen Virol* 21(1):135-143, 1973.

UV irradiation of SH-RP cells, a lymphoblastoid cell line carrying Epstein-Barr virus, which was derived from a patient with acute myeloid leukemia, retarded cell growth but enhanced the proportion of cells containing virus-capsid antigen (VCA) almost fourfold as demonstrated by indirect immunofluorescence. The effects of UV irradiation on SH-RP cultures were reversible under normal conditions of medium replenishment every four days. Starving the cultures did not modify these effects. The reason for the enhancement of VCA immunofluorescence by UV irradiation is not known. It is possible that there is relative protection of virus-DNA and virus replication, in the presence of damage to the host cell following UV irradiation. Another possibility is that there is a difference in sensitivity of the VCA-positive and VCA-negative cells to UV light.

- 4398 STUDIES ON A VIROGENIC CLONE OF SV 40-TRANSFORMED RABBIT CELLS USING CELL FUSION AND *IN SITU* HYBRIDIZATION. (E.) Watkins, J. F. (Sir William Dunn Sch. Path., Oxford, England). *J Gen Virol* 21(1):69-81, 1973.

A clone of baby rabbit kidney cells transformed with SV 40 virus was found to be releasing virus at the rate of about one infectious unit/10⁴ cells/day. Virus antigen was detected in between one in 10³ and one in 10⁴ cells. *In situ* hybridization with radioactive SV40 complementary RNA revealed that about 3% of the transformed cells were producing virus DNA. When the transformed cells were fused with permissive cells and incubated in anti-SV40 serum between 2% and 8% of the heterokaryons formed produced SV40 virus. A model is proposed according to which the cells in this clone, and possibly other transformed clones, may exist in one of three states: (A) the commonest state, in which virus DNA is not replicable autonomously; (B) a state in which virus DNA can be replicated autonomously,

but late virus proteins are not made; and (C) a state in which complete virus can be synthesized. The similarity between the proportions of SV40-transformed baby rabbit kidney cells (SVBRK) showing *in situ* hybridization and the proportion of heterokaryons showing evidence of SV40 growth suggests that the SVBRK cells which gave rise to virus in heterokaryons were those which were synthesizing virus DNA before fusion, and since the proportion of positive heterokaryons did not increase with time, that it is only these cells which could produce virus in a heterokaryon.

4399 ROUS SARCOMA VIRUS-INDUCED TUMORS IN MARMOSETS: CHARACTERISTICS OF TUMOR CELLS AND HOST CELL-VIRUS INTERRELATIONSHIPS. (E.)

Marczynska, B. (Rush Med. Ctr., Chicago, Ill.), F. Deinhardt, J. Schulien, P. Tischendorf and R. D. Smith. *J Natl Cancer Inst* 51(4):1255-1274, 1973. Thirty-eight tumors were induced in white-lipped marmosets (*Saguinus nigricollis*, *S. fuscicollis*) by the Schmidt-Ruppin strain of Rous sarcoma virus (SR-RSV). They were analyzed for viral genome, virus-expressed antigen(s), chromosomal aberrations, and the production of acid mucopolysaccharides (AMPS). Tumors established in cell cultures had morphologic characteristics typical for Rous tumor cells of fowl and other mammalian species. The viral genome was demonstrated in 7 of 18 tumors by cocultivation with live, X-irradiated, or disrupted chicken cells. No group-specific antigen was detected by complement fixation tests (COFAL) in tumors examined, and only a few animals with a long latent period developed COFAL antibodies. Although no specific chromosomal aberrations accompanied marmoset SR-RSV-induced tumors, chromosomes were often broken and rearranged, which was attributed to inadequate cell nutrition in fast-growing and poorly vascularized tumors. Large quantities of AMPS were demonstrated in some tumors, and all tumors had some AMPS on the periphery of tumors infiltrating surrounding muscles. Infectious viruses isolated from marmoset SR-RSV-induced tumors had envelope antigens similar to either subgroup A or D of avian sarcoma-leukosis viruses, as did the original SR-RSV used for marmoset inoculation.

4400 TUMORIGENICITY AND ANTIGENICITY OF MOUSE CELLS INFECTED WITH SIMIAN VIRUS 40. I. RELATIONSHIP OF GROWTH *IN VITRO* AND *IN VIVO* IN IMMUNOSUPPRESSED AND IMMUNOCOMPETENT RECIPIENTS. (E.) Wright, P. W. (Natl. Cancer Inst., Bethesda, Md.), H. S. Smith and J. McCoy. *J Natl Cancer Inst* 51(3):951-959, 1973.

The relationship between tumorigenicity and antigenicity of transformed cells in culture was examined. BALB/3T3 and several different classes of simian virus 40 (SV40) transformants derived from BALB/3T3 were tested for their ability to produce tumors in immunosuppressed recipients and to induce specific transplantation resistance to a transplantable SV40 tumor in immunocompetent recipients. SV40 tumors were defined by expression of the SV40 tumor (T)

antigen and the SV40 tumor-specific transplantation (TST) antigen. Only SV40-transformed cell lines expressing the TST antigen caused SV40 tumors. Although the "flat" transformants contained the T antigen in culture, they produced no SV40 tumors. Some cell lines lacking the SV40 TST antigen were tumorigenic, but the tumors were not SV40 derived. The SV40 TST antigen appeared to represent a unique marker for cells with the potential to become SV40 tumors.

4401 FELINE ONCORNAVIRUS-ASSOCIATED CELL MEMBRANE ANTIGEN. I. SEROLOGIC STUDIES WITH KITTENS EXPOSED TO CELL-FREE MATERIALS FROM VARIOUS FELINE FIBROSARCOMAS. (E.) Essex, M. (Harvard U. Sch. Public Hlth., Boston, Mass.) and S. P. Snyder. *J Natl Cancer Inst* 51(3):1007-1012, 1973.

Cell-free filtrates from three spontaneous feline fibrosarcomas and the Gardner-Arnstein feline sarcoma virus were each injected into four susceptible neonatal cats. Whole blood from a cat with a progressing sarcoma was also injected into one susceptible kitten. All 17 developed progressive malignant tumors. These kittens and their four nursing mothers were negative for antibody to the feline oncornavirus-associated cell membrane antigen (FOCMA). A fourth cell-free preparation was inoculated into two kittens. These kittens developed no tumors, but they and their nursing mother were positive for antibody to FOCMA. During the 23-wk examination period, the four previously FOCMA antibody-negative nursing mothers and four of five previously negative age-matched contact controls developed detectable humoral antibody titers. This was probably due to horizontal transmission of the feline oncornavirus. Because of these findings and previous evidence that FOCMA antibody-positive dams passively transmitted antibody to their kittens which then resisted the development of malignant tumors, it is recommended that contact control kittens or nursing mothers used in feline oncornavirus experiments not be returned to breeding colonies where kittens are produced for future experiments. It is suggested that as an additional precaution, cats of all ages used in such experiments be tested for FOCMA antibody before conclusions are drawn regarding the efficacy of any particular feline oncornavirus inoculum.

4402 IgM PRODUCTION IN RATS INFECTED WITH MOLONEY LEUKEMIA VIRUS. (E.) Cremer, N. E. (California State Dept. Public Hlth., Berkeley), D. O. N. Taylor, E. H. Lennette and S. J. Hagens. *J Natl Cancer Inst* 51(3):905-915, 1973.

Average serum IgM concentration of unstimulated rats infected with Moloney leukemia virus (MLV) was equal to or greater than that of unstimulated normal rats. After antigenic stimulation with *Salmonella typhi* "0" antigen, serum IgM of infected and normal rats increased, with the normal rats reaching a slightly but not significantly greater serum concentration. The average half life of IgM was 60-64 hr in both infected and normal rats. MLV antigen was present in 15-41% of the IgM-producing cells in

popliteal lymph nodes (PLN) of infected rats. The number of IgM-producing cells in the PLN of infected 6-wk-old rats was approximately equal to that in normal PLN but was reduced by 28-40% in 3- to 4-month-old infected rats. Serum IgM concentrations did not reflect this reduction, which suggested that PLN cells were not the major producers of IgM. Average antibody titers of infected rats after intravenous hyperimmunization with *S. typhi* "0" antigen did not differ significantly from normal. Since all of the infected rats were viremic, and many were overtly lymphomatous with thymus weights over 5 grams, it is concluded that MLV infection does not significantly depress IgM synthesis and that a normal functioning thymus is not essential for IgM production in these rats.

- 4403 HERPES SIMPLEX AND HERPES GENITALIS VIRUSES IN ETIOLOGY OF SOME HUMAN CANCERS. (E.) Sabin, A. B. (Natl. Cancer Inst., Frederick, Md.) and G. Tarro. *Proc Natl Acad Sci* 70(11): 3225-3229, 1973.

The sera of 57 people without cancer (including 18 persons with recurrent herpes simplex virus (HSV 1) or herpes genitalis virus (HSV 2) and 137 people with advanced cancer in 29 different body sites were tested for a specific reactivity with HSV nonvirion antigens. None of the sera from the subjects without cancer had nonvirion antibodies, nor did the sera of 81 of the cancer patients, including four with early malignant changes in the cervix uteri. However, all of the sera from the 56 patients with cancer of the following sites were positive for nonvirion antibodies: lip, mouth, oropharynx, nasopharynx, kidney, urinary bladder, prostate, cervix uteri, and vulva. Further, all seven patients with advanced cancers of the lip and oropharynx, areas commonly infected by HSV 1, reacted only with HSV 1 nonvirion antigens, while all 14 patients with advanced carcinoma of the cervix uteri or vulva, areas commonly infected by HSV 2, reacted with both HSV 1 and HSV 2 nonvirion antigens. Among the other positive patients, only two reacted with HSV 1 nonvirion antigens. The positive sera failed to react with cells taken at different times following high-multiplicity infection with the DNA vaccinia virus. Massive absorption of the positive sera with trypsinized, uninfected human embryonic kidney cells failed to remove, or lower the titer of the HSV 1 and HSV 2 nonvirion antibodies. The HSV 1 and HSV 2 appear to play an etiologic role in at least nine different types of human cancer.

- 4404 CELL-ASSOCIATED VIRIONS AND NUCLEOIDS OF A MURINE LEUKEMIA VIRUS. (E.) Panem, S. (Dept. Path., Pediatrics, U. Chicago, Ill.) and W. H. Kirsten. *J Natl Cancer Inst* 51(3):865-874, 1973.

Cell-associated nucleoids and virions were identified in the cytoplasm of established mouse cell lines infected with a murine leukemia virus. The buoyant density of the cell-associated virions ranged from

1.14-1.16 g/cm³; buoyant densities of cell-associated nucleoids were 1.22-1.24 g/cm³ after centrifugation through potassium citrate gradients. Cell-associated virions and nucleoids were isolated from potassium citrate gradient fractions that did not contain ribosomal RNAs. The cell-associated virions and nucleoids were distinguished from polysomes and mitochondria by their resistance to disruption by ethylenediaminetetraacetic acid (EDTA) and RNase. Cell-associated virions and nucleoids incorporated ³H-uridine within 10 min of exposure to label. Actinomycin D inhibited *de novo* incorporation of ³H-uridine into these structures within 20 min, but did not inhibit the maturation of previously labeled cell-associated virions and nucleoids into extracellular virions. Cell-associated virions contained 2 RNA species with sedimentation coefficients of 36 S and 20-24 S.

- 4405 FIBRINOLYSIS ASSOCIATED WITH ONCOGENIC TRANSFORMATION. REQUIREMENT OF PLASMINOGEN FOR CORRELATED CHANGES IN CELLULAR MORPHOLOGY, COLONY FORMATION IN AGAR, AND CELL MIGRATION. (E.) Ossowski, L. (Rockefeller U., New York, N.Y.), J. P. Quigley, G. M. Kellerman and E. Reich. *J Exp Med* 138(5):1056-1064, 1973.

Fetal bovine and dog serum were selectively freed of plasminogen by affinity chromatography. The resulting serum as well as native and reconstituted serum (obtained by the addition of purified plasminogen to the plasminogen-depleted serum) were used to examine the role of plasminogen in growth of normal and SV40-transformed hamster embryo fibroblasts in liquid medium; growth of SV40-transformed hamster embryo fibroblasts in soft agar; aggregation--a characteristic morphological change of SV40-transformed hamster cells; and migration of SV40-transformed and control 3T3 cells from a monolayer into a "wound". Exponential growth of both normal and transformed cells in liquid medium proceeded at the same rate in the presence or absence of plasminogen. In contrast, removal of plasminogen markedly depressed the plating efficiency of transformed cells in soft agar, eliminated their characteristic aggregation, and substantially reduced the extent of migration. Thus, the expression of these parameters of transformation is largely dependent on the activity of the fibrinolytic system.

- 4406 STUDIES ON MOUSE SARCOMA VIRUS. IV. THE ISOELECTRIC POINT OF THE GROUP-SPECIFIC ANTIGEN. (E.) Chuat, J. C. (Inst. Leukemia Res., Hopital Saint-Louis, Paris, France), C. Bernard, I. Laprevotte, C. Seban and M. Boiron. *Int J Cancer* 12(3):742-751, 1973.

Three, and in one instance four, classes of species-specific, group-specific (gs1) antigen-bearing molecules were separated by the isoelectric focusing of tween-ether disrupted Moloney strain mouse sarcoma virus (M-MSV) derived from a permanent producer rat cell line (78A1). The bulk of gs1 antigen, as measured by complement-fixation, was associated with a ³H-uridine labelled component characterized by an isoelectric point (ipH) of 5.9 and a sedimentation

coefficient of 2.7 $S_{w,20}$. Statistical evaluation showed the ipH value to be remarkably stable. Neither the ipH nor the 3H -uridine content was altered by ribonuclease (RNase); an ipH component found when nonpurified virus had been utilized was, however, sensitive to RNase. The other gsl antigen-bearing molecules were only minor components and included an RNase-resistant, ipH 5.5 component, frequently observed as a shoulder on the major peak, and an inconstant, poorly labelled component with a more variable ipH value of about 6.6. Two hypotheses regarding the nature of the association between gs antigen and RNA are proposed: the presence of 3H -uridine might result from mere attachment of nucleotides or oligonucleotides to an otherwise entirely proteic molecule; or the presence of uridine in the gs antigen might reflect some definite arrangement at the level of the substructure of the virion, i.e. a viral subunit. It is also possible that a gs antigen-bearing constituent is present in the viral envelope.

- 4407 EFFECTS OF 5-BROMO-2'-DEOXYURIDINE ON PRODUCTION OF GLOBIN MESSENGER RNA IN DIMETHYL SULFOXIDE-STIMULATED FRIEND LEUKEMIA CELLS. (E.) Preisler, H. D. (Mount Sinai Sch. Med., City U. New York, N. Y.), D. Housman, W. Scher and C. Friend. *Proc Natl Acad Sci USA* 70(10):2956-2959, 1973.

To determine the effect of 5-bromo-2'-deoxyuridine (BrdU) on the amount of globin mRNA present in Friend leukemia cells treated and not treated with dimethyl sulfoxide, molecular hybridization between total cell RNA and [3H]DNA complementary to mouse globin mRNA was used. Cells treated with BrdU and dimethyl sulfoxide had 70% less globin mRNA than cells treated with dimethyl sulfoxide alone. The size and base sequence of the residual globin mRNA in the cultures treated with BrdU and dimethyl sulfoxide were unaltered. Cells treated with BrdU alone contained slightly more globin mRNA than did the untreated controls, suggesting that BrdU may have a dual effect in transcription of messenger RNA.

- 4408 POLYOMA PSEUDOVIRIONS. II. INFLUENCE OF HOST CELL ON PSEDOVIRUS PRODUCTION. (E.) Yelton, D. B. (U. Maryland Sch. Med., Baltimore) and H. V. Aposhian. *J Virol* 12(5):1065-1071, 1973.

Polyoma and pseudovirus production by three infected mouse cell types was determined by DNA-DNA hybridization of purified radioactive particles and by sedimentation in alkaline sucrose gradients. The type of host cell influenced the relative amounts of pseudovirions and polyoma virions produced. The infection of primary mouse embryo cells resulted in the production of particles that were predominantly pseudovirions. Infection of baby mouse kidney or 3T3D cells yielded mainly infectious polyoma virus. The length of time that infection was allowed to continue also affected the amount of pseudovirions relative to polyoma virions. The longer the viral infection was allowed to proceed, the greater the quantity of pseudovirions produced. Pseudovirion

production could be correlated with the fragmentation of host cell DNA to a size of approximately 3×10^6 daltons. The fragmentation of host cell DNA was much more extensive in primary mouse embryo cells than in the other cell types.

- 4409 CHARACTERIZATION OF GUINEA-PIG EMBRYO CELLS TRANSFORMED BY KIRSTEN MOUSE SARCOMA VIRUS. (E.) Rhim, J. S. (Dept. Virus Res., Microbiol. Associates, Inc., Bethesda, Md.), F. G. Duh, H. Y. Cho and R. G. Huebner. *Int J Cancer* 12(3):589-601, 1973.

Guinea-pig embryo cells were transformed *in vitro* by the Kirsten strain of mouse sarcoma virus (Ki-MSV), but not by the Moloney or Harvey isolates of MSV. The transformed cells released infectious virus continuously and produced high titers of group-specific, complement-fixing antigen characteristic of the murine leukemia-sarcoma virus complex. The foci of the transformed cells were similar in appearance to those obtained with Ki-MSV in mouse and rat cells. The transformed cells produced RNA-dependent DNA polymerase and type-C virus particles with a density of approximately 1.15 g/ml in sucrose gradients by 3H -uridine labelling. The transformed cells from one line produced tumors when transplanted into newborn guinea-pigs. A number of focus-derived clonal lines and "normal cells" derived from infected cells were isolated and characterized. All the focus-derived lines were MSV producers. The Ki-MSV grown in guinea-pig cells replicated efficiently in guinea-pig and NRK cells but very poorly in mouse cells. A non-cytopathic type-C virus-producing line (clone No. 2) was isolated. A non-focus-forming virus grown in guinea-pig embryo cells (clone No. 2) rescued infectious MSV by direct cocultivation with Ki-MSV non-producer NRK cells. The rescued MSV virus was neutralized by Ki-MSV antiserum and produced foci readily in mouse, rat and guinea-pig cells. Further investigation revealed that Ki-MSV replicates and transforms cultures of canine embryo, rabbit kidney, pig kidney, feline embryo, and bovine embryonic kidney; these morphologically altered cells contained both infectious virus and gs antigen for viruses of murine sarcoma-leukemia complex.

- 4410 VIRAL-RELATED DNA SEQUENCES BEFORE AND AFTER TRANSFORMATION BY RNA TUMOR VIRUSES. (E.) Goodman, N. C. (Coll. Physicians Surgeons, Columbia U., New York, N.Y.), R. M. Ruprecht, R. W. Sweet, R. Massey, F. Deinhardt and S. Spiegelman. *Int J Cancer* 12(3):752-760, 1973.

A fibroblastic cell line (HF) was derived from a plasma clot culture of a skin biopsy from a 3-month-old marmoset and was subcultured every 7 to 10 days. At the sixth subculture, the cells were infected with either Rous sarcoma virus (SR-RSV), feline sarcoma virus (ST-FeSV), or simian sarcoma virus type 1 (SSV-1). The HF cells transformed by RSV and FeSV possessed the corresponding viral-specific DNA sequences (supporting the proviral hypothesis), while normal and SSV-1-transformed fibroblasts showed

only limited evidence of avian or feline viral sequences. These results suggest that the hybridization of DNA probes of RNA tumor viruses with normal cells can aid in assigning the host taxonomic position of newly isolated RNA tumor viruses.

- 4411 INTEGRATION OF DEOXYRIBONUCLEIC ACID SPECIFIC FOR ROUS SARCOMA VIRUS AFTER INFECTED OF PERMISSIVE AND NONPERMISSIVE HOSTS. (E.) Varmus, H. E. (Dept. Microbiol., U. California, San Francisco), P. K. Vogt and J. M. Bishop. *Proc Natl Acad Sci* 70(11):3067-3071, 1973.

A relatively simple, efficient, and stringent technique for studying the integration of DNA and RNA tumor virus genomes is presented. The technique involves testing the DNA in networks for virus-specific nucleotide sequences by molecular hybridization. The finding of virus-specific DNA in networks demonstrates covalent linkage of viral DNA to strands of cell DNA containing repeated sequences, and thus its integration into the host genome. This approach was used to study Rous sarcoma virus (RSV)-specific DNA in duck embryo fibroblasts and mammalian cells, particularly BALB/c 3T3 cells, following infection by RSV. In both cell types, neither of which normally contains nucleotide sequences specific for RSV, transformation by the virus resulted in the appearance of RSV-specific DNA, which was covalently integrated into strands of host-cell DNA containing reiterated sequences. The viral DNA appeared in the cells soon after infection, presumably as a product of the intracellular activity of virus-associated RNA-directed DNA polymerase. The assay therefore constitutes an assay for *in vivo* polymerase activity and may permit detailed analysis of the mechanism of RNA-directed DNA synthesis in the host cell. A potential limitation of the technique resides in the possibility that viral DNA might be integrated specifically into an uncommon region of the cell DNA that is devoid of reiterated sequences and does not form networks; in this case, integrated viral DNA would not be present in the networks. It is concluded that the genomes of both RNA and DNA tumor viruses are integrated into host-cell DNA, although the sites and mechanism for integration are unknown.

- 4412 EVIDENCE FOR HELPER INDEPENDENT MURINE SARCOMA VIRUS. I. SEGREGATION OF REPLICATION-DEFECTIVE AND TRANSFORMATION-DEFECTIVE VIRUSES. (E.) Ball, J. K. (Cancer Res. Lab., U. Western Ontario, London, Canada), J. A. McCarter and S. M. Sunderland. *Virology* 56(1):268-284, 1973.

Simultaneous assay for both murine sarcoma virus (MuSV) and murine leukemia virus (MuLV) was performed using a newly developed technique. Multiple rounds of infection were avoided by infecting cells in suspension, plating them sparsely, and allowing them to grow into colonies. XC cells were added to detect which colonies were producing leukemia virus. When cells were infected with the Moloney sarcoma-leukemia virus (M-MuSV (MuLV)), four types of colonies were

seen: (1) morphologically normal with syncytia (XC+) or (2) without syncytia (XC-), (3) morphologically transformed with no syncytia, (4) transformed with syncytia. The proportions infected by MuSV (transformed cells) or by MuLV (XC+) conformed to Poisson's distribution, and this allowed the calculation of the titers of MuSV and MuLV. Clones of chronically infected cells could readily be isolated. A clone of transformed cells called G8 was derived from JLS-V9 cells infected with M-MuSV (MuLV). The cells produced no MuLV detectable by cocultivation with XC cells, but they did produce sarcoma virus detected by the production of sarcomas in mice and morphological transformation of several lines of mouse cells in culture. The virus had a density of 1.16 g/cm³. The kinetics of focus formation were one-hit when assayed by the conventional assay. Virus picked from most (32/38) of these foci consisted of a mixture of sarcoma virus and leukemia virus but some (4/38) foci were found that produced sarcoma virus alone (presumably "competent" sarcoma virus, i.e., helper-independent). The presumed "competent" sarcoma virus was carried through four successive passages and each time, most of the foci were found to contain both MuSV and MuLV, but some produced MuSV only. In contrast, the original, chronically infected G8 cells did not release detectable MuLV through more than 30 passages. Leukemia virus or defective sarcoma virus segregated from the competent MuSV with low and equal frequencies only when new mouse cells were infected. No evidence was found for the presence of a helper virus in excess of the concentration of sarcoma virus and competence appears to be a property of the virion itself. In the search for a helper virus, a form of MuSV was found that did not morphologically transform the cells it infected, nor was it produced by them, but both transformation and release of MuSV appeared on superinfection with MuLV.

- 4413 PROTEIN METABOLISM IN SV40-INFECTED CELLS. (E.) Kiehn, E. D. (Dept. Biochem., U. Washington, Seattle). *Virology* 56(1):313-333, 1973.

Simian virus 40-induced alterations in protein metabolism were investigated in confluent monolayers of BS-C-1 monkey kidney cells. Viral-induced changes were observed which were specific for both the pre-replicative and the replicative phases of infection. The prereplicative phase, a viral-induced stimulation of protein synthesis, was observed at about 10-12 hr; by 20 hr after infection the rate of protein synthesis had increased to more than 20% above that of mock-infected cells. Gel electrophoresis of the proteins synthesized during the prereplicative period did not reveal any differences from control cells in any cell fraction except the cytosol. In this fraction several new species were observed, but together they did not constitute more than 1% of the overall rate of protein synthesis. An inhibition of labeling of one, and possibly two, host proteins was also discerned in the cytosol. Each of the polypeptide changes just described was also observed in infected cells which had been treated with cytosine arabinoside. This confirms the assignment of these effects to the early phase of infection, as

it has been well-established that cytosine arabinoside blocks viral DNA synthesis and all late viral functions, but has little effect on early events. During the replicative period the rate of protein synthesis continued to increase to more than 50% above that of mock-infected cells. Starting at 20 hr postinfection, proteins destined for the nucleus were synthesized at higher relative rates than those of cytoplasmic fractions. As a consequence, a very large progressive accumulation of protein in the nucleus began at about 40 hr after infection. Much of this increased nuclear mass could be accounted for as progeny virus particles found in the membranous fraction of sonicated nuclei. Gel electrophoresis of newly-made proteins late in infection showed that the two major viral structural proteins constituted about 20% of cellular protein synthesis. There was an additional viral-induced nonstructural polypeptide in the cytosol which constituted less than 1% of the newly-made proteins. As expected, these three new proteins were not synthesized in infected cells which had been treated with cytosine arabinoside. It was also observed that the labeling of the major host protein in the nuclear membrane was inhibited during the late phase of infection.

4414 ISOLATION OF C-TYPE RNA-VIRUSES FROM STABLE CELL CULTURES OF HUMAN TUMOURS.

(E.) Zhdanov, V. M. (Inst. Exp. Clin. Oncol., Acad. Med. Sci., Moscow, USSR), N. P. Mazurenko, L. S. Yakovleva, G. N. Trushinskaya and G. K. Gogichadze. *Neoplasma* 20(5):539-543, 1973.

The isolation of oncogenic RNA viruses from two cancerous (ovary and stomach), two sarcomatous (angiosarcomas), and one embryonic (muscle-epithelial) human cell line is discussed. All the cultures were contaminated with mycoplasmas which had density 1.2 to 1.22 g/cu cm in the sucrose gradients. Following treatment of the cultures with mitomycin C, three of five started production of mature and immature virions of C-type with a density of 1.16 to 1.17 g/cu cm; the production of the mycoplasmas simultaneously ceased. These phenomena were observed in all cultures of epithelial origin and did not take place in the sarcomatous cultures. In cultures treated with actinomycin D, no virion structures were observed. The virions had high molecular weight RNA with sedimentation coefficients 67-72S and slower sedimentating components. The purified virions showed reverse transcriptase activity with specific activity to DNA synthesis in the presence of artificial template (dT)10p(A). Thus the viruses had the characteristic features of C-type RNA-containing viruses.

4415 INFLUENCE OF INTERFERON ON VIRUS PARTICLE FORMATION IN DIFFERENT ONCORNAVIRUS CARRIER CELL LINES. (E.) Billiau, A. (Rega Inst., U. Leuven, Belgium), H. Sobis and P. de Somer. *Int J Cancer* 12(3):646-653, 1973.

The short-term effect of mouse interferon and viral functions in the following cell lines was studied:

cells producing infectious C-type particles, either Kirsten MSV in MO-P cells, or Rauscher-MLV in JLSV5 cells; S+L- cells carrying Moloney-MSV in a rescuable form and releasing noninfectious C-type particles; and MO4 cells carrying Kirsten-MSV in a rescuable form and producing intracisternal, non-infectious A-type particles. In the infectious virus-producing lines (MO-P and JLSV5), interferon caused a dose-dependent decrease in virus production as measured by the uridine incorporation technique. In S+L- cells, the uridine incorporation profile was not altered by exposure to interferon, although these results do not indicate whether the replication of the S+L- virus occurs through an interferon-sensitive mechanism. The MO4 cells displayed normal sensitivity to interferon when monitored by a vesicular stomatitis virus (VSV) challenge. Exposure to interferon did not prevent the increase in the type-A particle count induced by sequential exposure to bromodeoxyuridine and dimethylsulfoxide. Thus, the noninfectious A-type particles are synthesized by a different mechanism than that by which infectious C-type viruses are made, in that they escape the antiviral action of exogenous interferon.

4416 DNA OF EPSTEIN-BARR VIRUS DETECTED IN TISSUE OF BURKITT'S LYMPHOMA AND NASOPHARYNGEAL CARCINOMA. (E.) Nonoyama, M. (U. North Carolina Sch. Med., Chapel Hill), C. H. Huang, J. S. Pagano, G. Klein and S. Singh. *Proc Natl Acad Sci* 70(11):3265-3268, 1973.

The cRNA-DNA membrane hybridization method was used to determine the quantitative relationship between the viral DNA in Epstein-Barr virus (EBV)-carrying cells and Burkitt's lymphoma, nasopharyngeal carcinoma and 24 other tumors from patients in the same region of Kenya. Twenty-two of the 23 Burkitt's lymphomas were positive for EBV DNA, containing viral DNA in genome equivalent amounts from 4 to 113; 18 of the 23 nasopharyngeal carcinomas contained detectable EBV DNA; and 5 of the 24 other tumors (melanoma of the nose, carcinoma of the antrum, reticulum cell sarcoma, and two adenocarcinomas of the maxilla and mandible) were also positive for EBV DNA. The single case of Burkitt's lymphoma without detectable EBV DNA showed less than one genome equivalent per cell. The antibody titers of the Burkitt's lymphoma and nasopharyngeal carcinoma patients to virus capsid antigen was generally high, as were those of three of the other tumor cases which had been positive for EBV DNA. These data support the contention that EBV causes Burkitt's lymphoma.

4417 EVIDENCE FOR A HUMAN ONCOGENIC VIRUS. (E.) Willems, D. (St. Barbara Acad. Hosp., U. Leuven, Belgium), A. Billiau, H. Sobis and J. C. Mulier. *Acta Orthop Belg* 39(4):772-776, 1973.

Electron microscopic studies revealed the presence of C-type particles within the cytoplasm and budding from the surface of formalin-fixed tissue from an invasive human desmoid tumor removed by surgery. Nuclei of

these cells were free of particles. Examination of cells cultured from the tumor at the time of removal and in their sixth passage failed to show any sign of similar virus particles. These findings are considered as presumptive evidence of the existence of a human oncogenic virus.

- 4418 QUANTITATIVE STUDIES OF ONCORNAVIRUSES IN THIN SECTIONS. (E.) Miller, M. F. (U. Texas M. D. Anderson Hosp. Tumor Inst., Houston), P. T. Allen and L. Dmochowski. *J Gen Virol* 21:57-68, 1973.

A thin sectioning procedure was used to detect and enumerate oncornavirus particles in tumor-cell culture fluids, tumor homogenates, mouse blood plasma, mouse and human milk specimens, and density gradient fractions of these specimens. Oncornaviruses studied included mouse mammary tumor, murine sarcoma, ESP-1, and RD-114 viruses. In this procedure particles were sedimented on to small membrane filter discs in the ultracentrifuge using inexpensive commercially available adapters and tubes. Particles in cross sections of the disc were counted and their total number determined by relating the effective surface area of a field to the surface area of a field to the surface area of the entire membrane disc. Particles may be reliably identified and counted in preparations containing predominately cellular debris. Linear dose response plots were obtained in serial dilution experiments using vaccinia virus, adenovirus type 2, and murine sarcoma virus, demonstrating the reliability of the procedure and its wide applicability. The counts obtained for adenovirus and vaccinia virus preparations were comparable to counts obtained for the same preparations in other laboratories by established methods. Statistically reliable counts have been obtained using sample volumes of 0.01 ml or less. Virus particles of types other than those reported here could be quantitated by this procedure. The effective section thickness would have to be adjusted in accordance with virus particle diameter, actual section thickness, and the fraction of a particle required in a section for recognition.

- 4419 A BIOCHEMICAL CHANGE ASSOCIATED WITH NEOPLASTIC TRANSFORMATION DURING REVERSION AND BACK REVERSION OF A SV40 INDUCED TUMOR CELL LINE. (E.) Lallier, R. (U. Sherbrooke, Quebec, Canada), V. N. Nigam and C. Brailovsky. *Proc Am Assoc Cancer Res* 14(March):124, 1973.

- 4420 INITIAL STUDIES ON VIRUS ISOLATION FROM CANINE MILK SAMPLES. (E.) Manning, J. S. (U. California, Davis) and R. C. Guzman. *Proc Am Assoc Cancer Res* 14(March):115, 1973.

- 4421 CHROMATOGRAPHIC ANALYSIS OF 70 S RNA EXTRACTED FROM A MURINE ONCORNA VIRUS. (Fr.) Hampe, A. (St. Louis Hosp., Paris, France), M. E. Eladari, M. Gobet and M. Boiron. *C R Acad Sci [D] (Paris)* 276(13):2093-2095, 1973.

- 4422 VIRUS-LIKE PARTICLES IN THE BLOOD OF PATIENTS WITH LEUKEMIA. (Rus.) Shekolodkin, V. F. (No affiliation), F. L. Kiselev, T. I. Tikhonenko, I. Z. Zaretskii, M. T. Ivanov, A. F. Bykovskii, A. R. Zlatkina, G. V. Kruglova and Iu. L. Milevskaya. *Vestn Akad Med Nauk SSSR* (4):6-10, 1973.

- 4423 "REVERSION" OF A LESS TUMORIGENIC CYT MUTANT OF ADENOVIRUS 12 IN INDUCTION OF THE CELL SURFACE CHANGE. (E.) Yamamoto, H. (Nat'l. Inst. Hlth., Tokyo, Japan) and H. Shimojo. *J Virol* 12(2):413-414, 1973.

- 4424 EFFECT OF TEMPERATURE ON THE STRUCTURE OF LIGHT 8S RNA PRESENT IN MURINE SARCOMA VIRUS (MOLONEY STRAIN). (Fr.) Emanoil-Ravicovitch, R. (St. Louis Hosp., Paris, France), M. Bazilier, J. Robin and C. Jacques Larsen. *C R Acad Sci (Paris)* 277(6):617-620, 1973.

- 4425 ISOLATION OF A FELINE SARCOMA-INDUCING VIRUS FROM A SPONTANEOUS FIBROSARCOMA OF A CAT: STUDY OF THE SARCOMA-INDUCING POWER *IN VIVO*. (Fr.) Irgens, K. (Nat'l. Sch. Vet. Med. Maisons-Alfort, France), M. Wyers, A. Moraillon, A. Parodi and V. Fortuny. *C R Acad Sci [D] (Paris)* 276(11):1783-1786, 1973.

- 4426 EVIDENCE SUGGESTING THAT 'EARLY' VIRUS-SPECIFIC RNA MAY CONTAIN INFORMATION NECESSARY FOR SV40-INDUCED CHROMOSOME REPLICATION AND MITOSIS. (E.) May, E. (Dept. Molec. Biol., U. Geneva, Switzerland), P. May, and R. Weil. *Experientia* 29(6):776, 1973.

- 4427 SEQUENCE OF POLYOMA VIRUS INDUCED CHROMOSOMAL DNA SYNTHESIS IN PRIMARY MOUSE KIDNEY CELL CULTURES. (E.) Farrell, P. (Dept. Molec. Biol., U. Geneva, Genève), and R. Weil. *Experientia* 29(6):770, 1973.

- 4428 UNUSUAL INTRANUCLEAR TUBULAR STRUCTURES ASSOCIATED WITH THE MATURATION OF *HERPES-VIRUS SAIMIRI* IN MONKEY KIDNEY CELL CULTURES. (E.) Morgan, D. G. (Dept. Path., U. Bristol, England), B. G. Achong, and M. A. Epstein. *Br J Cancer* 27(6):434-440, 1973.

- 4429 INHIBITION OF MALIGNANT CELL INVASION *IN VITRO* BY A PROTEINASE INHIBITOR. (E.) Latner, A. L. (Royal Victoria Infirmary, Newcastle upon Tyne, England), E. Longstaff and K. Pradhan. *Br J Cancer* 27(6):460-464, 1973.

- 4430 HEMOPOIETIC STEM CELLS IN MURINE VIRUS-INDUCED LEUKEMIA. I. SPLEEN COLONY ASSAYS IN CBA MICE AFTER RAUSCHER VIRUS INFECTION. (Ger.) Seidel, H.-J. (Ctr. Basic Clinical Res., U. Ulm, Germany). *Z Krebsforsch* 79(2):123-134, 1973.
- 4431 GENITAL HERPES INFECTION. (LITERATURE REVIEW). (Rus.) Pukhner, A. F. (No affiliation), V. I. Kozlova and I. M. Misiurev. *Akush Ginekol (Mosk)* (3):3-6, 1973.
- 4432 INHIBITORY ACTION OF VITAMIN A ON A MURINE SARCOMA. (E.) Seifter, E. (A. Einstein Coll. Med., Yeshiva U., New York, N.Y.), M. Zisblatt, N. Levine and G. Rettura. *Life Sci* 13(7):945-952, 1973.
- 4433 REPLICATION OF RAUSCHER VIRUS *IN VIVO* IN RESISTANT MICE WITH SIMPLE AND MIXED MYCOPLASMA-VIRUS INFECTIONS. (Rus.) Postnikova, Z. A. (N. F. Gamaleia Inst. Epidemiol. Microbiol., Moscow, USSR), T. D. Morgunova, I. V. Rakovskaia and G. Ia. Kagan. *Vestn Akad Med Nauk SSSR* (2):75-79, 1973.
- 4434 FORMATION OF AVIAN ONCORNAVIRUS PROTEINS: IDENTIFICATION OF A HIGH MOLECULAR WEIGHT PRECURSOR. (E.) Vogt, V. M. (Swiss Inst. Experimental Cancer Res., Lausanne, Switzerland), R. Eisenman and H. Diggelmann. *Experientia* 29(6):780, 1973.
- 4435 ISOLATION OF AN IMMUNOGENIC POLYPEPTIDE AFTER LIMITED TRYPSIN HYDROLYSIS OF MURINE LEUKEMIA VIRUS GROUP-SPECIFIC ANTIGEN. (E.) Davis, J. (Flow Lab., Inc., Rockville, Md.), R. V. Gilden and S. Oroszlan. *Virology* 56(1):411-415, 1973.
- 4436 A CIRCULAR DNA-PROTEIN COMPLEX FROM ADENOVIRUSES. (E.) Robinson, A. J. (Sch. Med. Res., Australian Natl. U., Canberra), H. B. Younghusband and A. J. D. Bellett. *Virology* 56(1):54-69, 1973.
- 4437 DIFFUSION COEFFICIENTS AND HYDRODYNAMIC RADII OF THREE SPHERICAL RNA VIRUSES BY LASER LIGHT SCATTERING. (E.) Harvey, J. D. (U. Auckland, New Zealand). *Virology* 56(1):365-368, 1973.
- 4438 XC CELL CYTOPATHOGENICITY AS AN ASSAY FOR MURINE MYELOMA C-TYPE VIRUS. (E.) Volkman, L. E. (Mayo Fdn., Rochester, Minn.) and R. G. Krueger. *J Natl Cancer Inst* 51(4):1205-1210, 1973.
- 4439 DIETHYLAMINOETHYL-DEXTRAN AND UPTAKE OF NUCLEIC ACIDS BY MAMMALIAN CELLS. (E.) Borenfreund, E. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), M. Steinglass, G. C. Korngold and A. Bendich. *J Natl Cancer Inst* 51(4):1391-1392, 1973.
- 4440 THE EFFECT OF NUCLEIC ACID FROM CHICK ROUS SARCOMA ON EMBRYONAL HUMAN AND CHICK CELLS *IN VITRO*. (Rus.) Kuznetsov, O. K. (USSR Ministry Publ. Hlth., Leningrad), A. I. Zhudina, V. P. Goncharova and A. M. Dyadjkova. *Vopr Onkol* 19(9):67-74, 1973.
- 4441 STUDIES ON THE PROBLEM OF THE VIRUS ETIOLOGY OF TUMORS. (Rus.) Graffi, A. (Inst. Cancer Res., East German Acad. Sci., Berlin). *Vopr Onkol* 19(9):54-60, 1973.
- 4442 INFECTIOUS HERPESVIRUS DNA. (E.) Graham, F. L. (Lab. Physiological Chem., St. U. Leiden, Netherlands), G. Veldhuisen and N. M. Wilkie. *Nature [New Biol]* 245(148):265-266, 1973.
- 4443 CONTROLLED EXPRESSION OF SV40 GENOME. (E.) Naha, P. M. (Natl. Inst. Med. Res., London, England). *Nature [New Biol]* 245(148):266-268, 1973.
- 4444 PROTECTIVE EFFECT OF THE DOUBLE-STRANDED POLYRIBONUCLEOTIDE, POLYINOSINIC-POLYCYTIDYLIC ACID, AGAINST RAT ERYTHROBLASTOSIS INDUCED BY MURINE ERYTHROBLASTOSIS VIRUS. (E.) Slamon, D. J. (Dept. Anatomy, U. Chicago, Ill.). *J Natl Cancer Inst* 51(3):851-863, 1973.
- 4445 DIFFERENCE IN HEAT STABILITY OF ANTIGENS ASSOCIATED WITH EPSTEIN-BARR VIRUS, DEMONSTRATED BY IMMUNODIFFUSION. (E.) Demissie, A. (Central Microbiol. Lab., Stockholm Cty. Council, Sweden). *J Natl Cancer Inst* 51(3):751-760, 1973.
- 4446 REASSOCIATION KINETICS FOR EPSTEIN-BARR VIRUS DNA: NONHOMOLOGY TO MAMMALIAN DNA AND HOMOLOGY OF VIRAL DNA IN VARIOUS DISEASES. (E.) Kawai, Y. (Sch. Med., U. North Carolina, Chapel Hill), M. Nonoyama and J. S. Pagano. *J Virol* 12(5):1006-1012, 1973.
- 4447 DIFFERENTIAL RESPONSE OF RAT TOOTH GERMS TO A MURINE SARCOMA VIRUS IN CELL CULTURE AND SYNGENEIC TRANSPLANTS. (E.) Schwartz, S. A. (Dept. Path., U. Chicago, Ill.) and W. H. Kirsten. *J Natl Cancer Soc* 51(4):1163-1169, 1973.
- 4448 HUMAN TUMOR CELL MIGRATION. (E.) Cochran, A. J. (Western Infirm., Glasgow, Scotland), R. Kiessling, E. Klein, P. Gunven and A. K. Foulis. *J Natl Cancer Inst* 51(4):1109-1111, 1973.
- 4449 RELATIONSHIP BETWEEN POST-TRANSCRIPTIONAL ADENYLATION OF HERPES VIRUS RNA AND MESSENGER RNA ABUNDANCE. (E.) Silverstein, S. (Dept. Microbiol. Biophys., U. Chicago, Illinois), S. L. Bachenheimer, N. Frenkel, and B. Roizman. *Proc Natl Acad Sci* 70(7):2101-2104, 1973.

- 4450 EPSTEIN-BARR VIRUS AND INFECTIOUS MONONUCLEOSIS. (E.) Royston, I. (U.S. Publ. Hlth. Service, Bethesda, Md.). *Lancet* (7838):1152, 1973.
- 4451 EVIDENCE THAT THE POLYADENYLIC ACID SEGMENT OF "35S" RNA OF AVIAN MYELOBLASTOSIS VIRUS IS LOCATED AT THE 3'-OH TERMINUS. (E.) Stephenson, M. L. (Massachusetts Gen. Hosp., Boston), J. F. Scott and P. C. Zamecnik. *Biochem Biophys Res Commun* 55(1):8-16, 1973.
- 4452 KAPOSI'S SARCOMA. TISSUE CULTURE STUDIES. VIROLOGICAL AND IMMUNOLOGICAL STUDIES. (E.) Giraldo, G. (St. Louis Hosp., Paris, France), E. Beth, F. Haguenau, G. Noury, A. Puissant and J. M. Huraux. *Ann Dermatol Syphiligr* 100(3):283-284, 1973.
- 4453 INTRACISTERNAL VIRAL C AND A PARTICLES IN SPONTANEOUS LUNG TUMORS OCCURRING IN BALB/c/Cb/Se MICE. (It.) Bucciarelli, E. (Div. Cancer Res., U. Perugia, Italy). *Lab Anat Patol Perugia* 32:109-120, 1972.
- 4454 STUDY OF MAREK'S LYMPHOMA. COMMUNICATION I. VIRUS ISOLATION, SOME IMMUNOLOGICAL AND PATHOMORPHOLOGICAL INVESTIGATIONS. (Rus.) Mazurenko, N. P. (Inst. Exp. Clin. Oncol., Moscow, USSR), Z. I. Merekalova, L. S. Yakovleva, V. N. Stepina, V. N. Vinogradov, I. Yu. Chernyakhovskaya, N. K. Gunenkova, A. I. Pavlovskaya, N. F. Grinenko, L. V. Shershulskaya, V. E. Gurtsevich and Yu. N. Zueva. *Vestn Akad Med Nauk SSSR* 27(10):11-20, 1972.
- 4455 GENETIC STUDIES OF TEMPERATURE-SENSITIVE MUTANTS OF MOLONEY-MURINE LEUKEMIA VIRUS. (E.) Wong, P. K. Y. (Cancer Res. Lab., U. of Western London, Ontario, Canada) and J. A. McCarter. *Virology* 53(2):319-326, 1973.
- 4456 EXPERIMENTAL HUMAN REINFECTION WITH HERPES SIMPLEX VIRUS. (E.) Blank, H. (U. Miami, Sch. Med., Fla.) and H. G. Haines. *J Invest Dermatol* 61(4):223-225, 1973.
- 4457 ULTRASTRUCTURE OF SARCOMA 180 CELLS IN MICE TREATED WITH TESTOSTERONE PROPIONATE. VIRUS-LIKE BODIES IN THE SARCOMATOUS CELL NUCLEUS. (E.) Miscalencu, D. (I. Cantacuzino Inst., Bucharest, Rumania), F. Mailat and M. D. Ionescu. *Anat Anz* 134(3):215-224, 1973.
- 4458 THE INTERACTION OF HERPES SIMPLEX VIRUS WITH CULTURES OF PERIPHERAL NERVOUS TISSUE: AN ELECTRON MICROSCOPIC STUDY. (E.) Hill, T. J. (Med. Sch., U. Walk, Bristol, England) and H. J. Field. *J Gen Virol* 21:123-133, 1973.
- 4459 ATTEMPTS TO DETECT MAMMARY TUMOR VIRUS IN THE SPERMATIC TRACT OF OLD BALB/C MICE: PRELIMINARY REPORT. (E.) Squartini, F. (Inst. Path. Anatomy, Histology, U. Pisa, Italy), E. Bucciarelli, R. Ribacchi and G. B. Bolis. *Lav Anat Patol Perugia* 32(3):97-102, 1972.
- 4460 BURKITT'S LYMPHOMA. A CLINICO-PATHOLOGICAL REVIEW OF IBADAN CASES. (E.) Osunkoya, B. O. (U. Coll. Hosp., Ibadan, Nigeria) and O. O. Ajayi. *Paediatrician* 1(4-5):261-272, 1972/1973.

See also:

- * (Rev): 4201, 4204, 4205, 4216, 4228, 4229, 4252
- * (Chem): 4273
- * (Immun): 4471, 4472, 4475, 4478, 4491, 4498, 4499, 4500, 4501, 4503
- * (Epid-Biom): 4550

- 4461 "B"-CELL STIMULATION OF ALLOGENEIC T-CELL PROLIFERATION IN MIXED LYMPHOCYTE CULTURES. (E.) Plate, J. M. D. (Harvard Med. Sch., Boston, Mass.) and I. F. C. McKenzie. *Nature [New Biol]* 245(147):247-249, 1973.

The types of cells interacting in mixtures of allogeneic lymphocytes in culture (mixed lymphocyte reaction: MLR) were studied through the use of two different antisera: one directed against thymus-derived lymphocytes (θ -positive cells), and the other directed against "B" lymphocytes (non- θ -bearing cells). "B" cells, that is those remaining after treatment of lymph node cell suspensions from female C57Bl/10 and B10.D2/n mice with anti- θ serum and complement (C') responded poorly in the MLR assay compared to cells treated with normal mouse serum and C'. This indicated that the T cell population accounted for the majority of proliferating cells. "B" cell-enriched preparations exhibited enhanced stimulation of proliferative response, while "B" cell preparations did not readily respond to allogeneic "B" cells. Elimination of "B" cells in serum depressed the ability of lymphocytes to stimulate but did not influence the level of response in the MLR, while treatment with anti- θ serum depressed responsiveness without reducing the ability to stimulate. These results demonstrate that T-derived lymphocytes are stimulated to proliferate by foreign "B" lymphocytes. Thus, it is mainly the interaction between allogeneic T and "B" lymphocytes that leads to the cell transformation observed in the MLR.

- 4462 CIRCULATING ANTIBODIES IN HUMAN CONNECTIVE TISSUE MALIGNANCY. (E.) Moore, M. (Robert Jones and Agnes Hunt Orthopedic Hosp., Oswestry, Shropshire, England) and L. A. Hughes. *Br J Cancer* 29(1):175-184, 1973.

In a comparative study, sera from patients with connective tissue tumors, various carcinomas and from individuals without malignancy were evaluated by indirect immunofluorescence (IF) for antibodies reactive with apparently specific antigens shared by sarcoma derived tissue culture cell lines; for antibodies by IF to two tissue autoantigens (nuclear antigen and smooth muscle antigen) on rat liver substrate; and for HL-A antibodies by microcytotoxicity against a panel of 22 lymphocyte preparations. Neoplasms from which successful tissue cultures were initiated for these experiments were taken from patients with osteosarcoma (7), chondrosarcoma (2), fibrosarcoma (3), rhabdomyosarcoma (1), Ewing's tumor (1), chordoma (1) and liposarcoma (1). Antibodies reactive with 11/16 cell lines originating from sarcomas were detected in 36% of all sarcoma sera tested, 12% carcinoma sera and 9% sera from controls. The incidence of antisarcoma antibody (ASA) was higher in the sera of sarcoma patients whose disease was in the primary phase (53%) compared with those in whom disease was advanced. A lower incidence (8%) of antibodies to nuclear antigen was detected in the sera of sarcoma patients compared with carcinoma patients (22%) and controls (23%), but the incidence of smooth muscle antibodies was higher (45%) compared with carcinomas and controls (36% and 35% resp.). HL-A antibodies

were present in 13% sarcoma sera, 36% carcinoma sera and 33% control sera. Evidence is presented to show that the various antibodies are distinct and that those reacting with sarcoma derived cell lines may be tumor-associated antibodies possibly related to a virus specified antigen.

- 4463 AUSTRALIA ANTIGEN IN PRIMARY LIVER CANCER: ITS RELATION WITH ALPHA-FETOPROTEIN. (Fr.) Bourgeaux, C. (Fac. Med., Dijon, France), C. Trepo, M. Bordes, P. Sizaret, F. Martin, R. Sananes, M. Sepetjian and C. Klepping. *Biol Gastroenterol (Paris)* 6(2):133-137, 1973.

Of 32 patients (27 men and 5 women) with primary liver cancer, 22 had positive tests for serum α -fetoprotein but only one had Australia antigen. All but one of these patients was of French origin; liver cancer was associated with cirrhosis in 12 of 16 patients autopsied. The patient with Australia antigen was a 72-yr-old male alcoholic with no history of viral hepatitis or blood transfusions, a positive test for α -fetoprotein, and advanced septal cirrhosis. In another group of 13 patients (11 men and 2 women), all of whom had positive tests for serum α -fetoprotein and a variety of diseases other than primary liver cancer, only 2 had Australia antigen. There were a 2-yr-old boy with a testicular dysembryoma who had received a blood transfusion and a patient with viral hepatitis. The incidence of viral hepatitis in these patients with primary liver cancer and positive serum α -fetoprotein tests is about the same as in the general population in France. These results differ from others obtained in some countries of Africa and Asia (Formosa, Uganda, and Senegal) where primary liver cancer is often associated with Australia antigen. Therefore, viral hepatitis does not appear to be an important factor in the etiology of primary liver cancer in France.

- 4464 GENETICALLY DETERMINED EFFECT OF TRANSPLANTED THYMUS IN A MILLIPORE CHAMBER ON THE DEVELOPMENT OF SYNGENEIC MAMMARY TUMORS. (Rus.) Videlets, I. Iu. (Inst. Cytol. Genetics, Novosibirsk, USSR), E. V. Gruntenko and D. K. Beliaev. *Dokl Akad Nauk SSSR* 211(2):465-467, 1973.

High-cancer C3H/He mice were thymectomized 5-6 days after birth and given i.p. implants of thymus from C3H/H3 (high-cancer), C57Bl/6J (low cancer) and A/HeJ (high-cancer) mice; thymus was sealed hermetically in millipore filters. One month after implantation mice were injected s.c. with a 20% suspension of mammary tumor cells from female C3H/He mice. The growth rate of mammary tumors was slower in thymectomized mice than in intact controls, but the tumor growth rate was faster in mice given i.p. thymus implants from C3H/He mice than in intact controls. This increased growth rate is attributed to a humoral factor produced by the thymus implants. The tumor growth rate was faster than control values in mice given thymus implants from A/HeJ mice and was slower than control values in mice given thymus implants from C57Bl/6J mice. These differences in

growth rates cannot be attributed to any antigenic difference between the thymus implant and the host. It is suggested that the humoral factor is produced by reticuloendothelial cells of the thymus.

- 4465 α -FETOPROTEIN AND AUSTRALIA ANTIGEN IN PRIMARY LIVER CANCER. (E.) Dammacco, F. (U. Bari Med. Sch., Italy), A. Miglietta, S. Antonaci and L. Bonomo. *Digestion* 9(1):41-48, 1973.

Sera from patients with neoplastic and non-neoplastic diseases, as well as from full-term newborns and pregnant women were examined for the presence of α -fetoprotein (α -FP) by counterimmunoelectrophoresis. α -FP was detected in primary hepatoma (59%) and gonadal teratoblastoma. Positive results were also obtained in a case of colonic carcinoma metastatic to the liver and in 100% of cord blood sera. Since counterimmunoelectrophoresis is approximately intermediate between double diffusion and radioimmunoassay in terms of degree of sensitivity, it was proposed that this technique should be employed routinely for clinical purposes. The frequency of the hepatitis-associated antigen (HBAg) was determined in the sera of hepatoma patients and of two control groups consisting of healthy blood donors and patients with malignant tumors other than hepatoma. HBAg was present in 1 (4.5%) of the 22 hepatoma sera. By comparison, it was detected in 1 (4%) of the 25 sera belonging to the neoplastic series and in 5 (1.9%) of 950 blood donors. It is suggested that primary hepatoma is not directly related to chronic hepatitis and/or presence of HBAg. When, however, peculiar relationships occur between host and the hepatitis virus, viral hepatitis may progress to liver-cell carcinoma through the intermediate stage of post-necrotic cirrhosis.

- 4466 ANTIGENIC AND MORPHOLOGIC PROPERTIES OF OVARIAN CARCINOMA. (E.) Ioachim, H. L. (Lenox Hill Hosp., New York, N.Y.), B. H. Dorsett, M. Sabbath, B. Andersson and H. R. K. Barber. *Gynecol Oncol* 1:130-142, 1973.

As part of a multilateral study of ovarian carcinoma comprising 68 cases, tumors representing all four major histologic types were explanted in tissue cultures, examined under the electron microscope, and used in immunologic assays. The ultrastructure was characterized by nuclear and nucleolar pleomorphism which correlated well with the degree of malignancy and tumor grading. Cytoplasmic organelles and intercellular junctions were abundant and fairly well differentiated even in ovarian carcinoma of higher grade and stage. Active processes of synthesis and secretion taking place in most of these tumors were suggested by the presence of a richly granular endoplasmic reticulum, dilated cisternae, and numerous secretory granules. All tumors were cultured *in vitro* and their morphology in light and electron microscopy was compared to that of the original tumors. They displayed a consistent pattern of growth after several months *in vitro* and numerous subcultures, which led to the conclusion that ovarian tumor cells in culture have preserved most of their salient features and are

representative of the original tumors from which they have been derived. The immunologic studies included cytotoxicity assays using the patient's own serum and were indicative of the presence of specific humoral antibodies. In other studies a specific antiserum was prepared in a heterologous system then concentrated, purified, and fractionated. Immunofluorescence and immunodiffusion assays revealed a high degree of specificity for this antiserum.

- 4467 ANTIGENS AND ANTIBODIES CIRCULATING IN THE BLOOD DURING THE EARLY STAGES OF EXPERIMENTAL HEPATOCARCINOGENESIS. (Rus.) Korosteleva, T. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR) and L. S. Potapenkova. *Vopr Onkol* 19(7):53-58, 1973.

Antigens and antibodies were investigated in sera and liver extracts from male C3HA mice fed 2 mg/day of o-aminoozotoluene (OAT) for as long as 100 days. By precipitation in gel with heterologous immune sera for OAT-azoproteins, an antigen that was not present in untreated controls was detected in sera and liver extracts from mice fed OAT for 60 and 100 days. Immuno-electrophoresis revealed that this antigen had a mobility in the α -globulin and albumin range. Circulating antibodies for OAT-haptenes were detected in all mice given OAT for 60 days or more. On electrophoresis, these antibodies had a mobility in the α -globulin and albumin range. These findings suggest that highly sensitive immunological techniques might be used for the early diagnosis of chemically-induced cancer in man by the detection of circulating antigens and antibodies produced in the autoimmune processes which occur in carcinogenesis.

- 4468 *IN VITRO* SPLENIC IgG SYNTHESIS IN HODGKIN'S DISEASE. (E.) Longmire, R. L. (Scripps Clin. Res. Fdn., La Jolla, Calif.), R. McMillan, R. Yelenosky, S. Armstrong, J. E. Lang and C. G. Craddock. *N Engl J Med* 289(15):763-767, 1973.

Experimental and clinical observations suggest increased lymphoid reactivity in Hodgkin's disease. Since thymus-dependent lymphocyte function is often depressed whereas serum antibody responses appear normal, *in vitro* splenic IgG synthesis was studied to assess B lymphocyte activity. Increased total splenic IgG synthesis occurred in 20 of 22 patients with Hodgkin's disease. Mean IgG production by uninvolved and lightly involved spleens of the patients was five and 11 times normal, while heavily involved spleens averaged twice normal levels. Unstimulated *in vitro* IgG synthesis by uninvolved and lightly involved spleens of patients was similar to smallpox vaccine-stimulated *in vitro* IgG synthesis by normal spleens, suggesting an *in vitro* response of the patients' spleens to prior *in vivo* antigenic challenge. The nature of the hypothetical antigen which served to challenge the patients' spleens *in vivo* is not known. When splenic-culture IgG of patients was incubated with homologous lymphocytes, highly significant IgG binding levels were noted. These data suggest that the spleen in Hodgkin's disease responds with a

humoral antibody to some antigen associated with lymphocytes.

- 4469 ACTIVE HOST RESISTANCE FOR TRANSPLANT SYNGENEIC C57BL LYMPHOMAS. (E.) Maruyama, Y. (U. Kentucky Med. Ctr., Lexington) and J. M. Feola. *Oncology* 28(1):52-62, 1973.

Tumor-induced immunity was studied in inbred C57BL mice inoculated with LSY lymphoma cells, a syngeneic line newly adapted for growth in this strain of mice. Host resistance could be induced with X-irradiated LSY lymphoma cells following a regimen of weekly i.m. inoculations for 3 wk. Under these conditions, the cell dose required to produce 50% tumor takes (TD₅₀) was increased about five-fold. A five-fold suppression in host resistance was achieved by a 400-R whole-body X-irradiation dose prior to challenge. Host resistance to LSA lymphoma cells, a line which was adapted to C57BL mice over a prolonged period, could be induced only following serial sublethal tumor challenge with small numbers of viable cells whereby the TD₅₀ was increased five- to ten-fold. Attempts to induce immunity with X-irradiated LSA cells were unsuccessful.

- 4470 ANTIBODY DEPENDENT CELLULAR IMMUNITY IN NEWBORN MICE. (E.) Ralph, P. (Salk Inst. Biol. Studies, San Diego, Calif.), I. Nakoinz and M. Cohn. *Nature [New Biol]* 245(144):157-158, 1973.

Sera prepared from BALB/c female mice immunized before mating with 3-6 weekly i.v. injections of sheep erythrocytes and from progeny of the immunized mother were tested for ability to effect antibody-dependent cellular immunity by addition to unimmunized adult spleen cells and chromium-labeled sheep erythrocytes. Maternal and newborn sera caused lysis of the target at final dilutions of 10⁻³ to 10⁻⁵. The lysis was specific, for normal postpartum or normal newborn sera were not effective, and the specific sera did not release chromium from a different target, TNP-horse erythrocytes. The presence of effector cells for antibody-dependent cellular immunity was also studied in mice of various ages. Liver and spleen cells of late-term fetuses and newborn mice had an activity comparable to or slightly higher than that of adult spleen cells. Newborn thymus and adult liver and thymus were relatively inactive. These studies indicate that newborn mice derive a maternal, antigen-specific factor, presumably antibody, which can direct the lysis of a target by an effector cell present in the fetus and newborn.

- 4471 RADIOIMMUNOASSAY FOR AVIAN C-TYPE VIRUS GROUP-SPECIFIC ANTIGEN: DETECTION IN NORMAL AND VIRUS-TRANSFORMED CELLS. (E.) Stephenson, J. R. (Natl. Cancer Inst., Bethesda, Md.), R. E. Wilsnack and S. A. Aaronson. *J Virol* 11(6):893-899, 1973.

A radioimmunoassay developed for detection of avian C-type virus (30,000 molecular wt) group-specific (gs) antigen is 10- to 1,000-fold more sensitive than immuno-

logical methods previously available. By the radioimmunoassay technique, normal chicken embryo cells, which have previously been classified as gs negative or weakly gs positive, contain clearly detectable amounts of gs antigen. The assay was used to study the effect of chemical induction and superinfection by mammalian C-type viruses on the expression of avian gs antigen in mammalian cells nonproductively transformed by avian sarcoma viruses. When treated with 5-iododeoxyuridine (IdU, µg/ml for 24 hr), BALB/3T3 and normal rat kidney cells transformed by the Schmidt-Rupin strain of Rous sarcoma virus showed a two- to three-fold increase in avian gs antigen. Although the amount of mouse gs antigen/mg of cell protein increased approximately 100-fold after superinfection with R-MuLV, there was no detectable increase in the expression of avian gs antigen. These findings indicate that IdU induces avian gs antigen expression in sarcoma-virus transformed cells independently of its effect on the endogenous mouse C-type virus.

- 4472 ANTIGENIC STRUCTURE OF MURINE MAMMARY TUMOUR VIRUSES. (E.) Daams, J. H. (Netherlands Cancer Inst., Amsterdam), P. Hageman, J. Calafat and P. Bentvelzen. *Eur J Cancer* 9(8):567-572, 1973.

Rabbit antisera against purified standard murine mammary tumor virus (MTV-S) precipitated the virions of four different virus strains in double immunodiffusion: MTV-S, -L, -P and -O. The sera detected eight soluble antigens of MTV-S and -O, but only five of -P and three of -L. The effect of trypsin, pepsin, HCl RNase, phospholipase C, and acridine orange on the appearance of precipitation lines of MTV-S antigens were investigated. The results indicate that two soluble trypsin-sensitive antigens are located inside the nucleoid. They seem to contain some RNA fragments which do not influence antigenicity. They are group specific. Three other soluble antigens are membrane-constituents. Phospholipase C seems to increase their reactivity in immunodiffusion. They all contain a protein moiety which is essential for antigenicity. Two of the membrane antigens are involved in immunoprecipitation of the intact virion; one of them being group-specific.

- 4473 DEREPRESSION OF ALLOANTIGENS IN MALIGNANCY. EVIDENCE FOR TUMOUR SUSCEPTIBILITY ALLOANTIGENS AND FOR POSSIBLE SELF-REACTIVITY OF LYMPHOID CELLS ACTIVE IN THE MICROCYTOTOXICITY ASSAY. (E.) Martin, W. J. (Natl. Cancer Inst., Bethesda, Maryland), E. Esber, W. G. Cotton and J. M. Rice. *Br J Cancer* 28(Suppl 1):48-61, 1973.

Inbred strains of mice were found to differ markedly in both susceptibility to the spontaneous development of malignant alveologenic lung tumors and the ease with which these tumors could be induced with chemical carcinogens administered to adult animals. Malignant lung tumors occurred in normal strain A mice but were very rare in normal C3Hf, DBA/2 and C57BL6 mice or in these mice treated as adults with the carcinogen 1-

ethyl-1-nitrosourea (ENU). Malignant tumors could, however, be induced in C3Hf mice exposed prenatally to ENU. Two transplacentally induced malignant lung tumors of C3Hf mice failed to grow when transplanted to normal C3Hf recipients but did grow progressively when transplanted into either (C3Hf x A) F₁ hybrid or C3H recipients. The tumors grew progressively in sublethally x-irradiated but otherwise untreated C3Hf mice. Immunization of C3Hf mice with either of the lung tumors, or with normal lung tissue of either A or C3H mice, induced a degree of radioresistant immunity such that tumor cells inoculated into immunized, sublethally x-irradiated mice, failed to grow progressively. Radioresistant immunity was not induced when C3Hf mice were immunized with lung tissue of DBA/2 or C57BL/6 mice. Lymphoid cells of (C3Hf x A) F₁ and C3H mice bearing transplanted C3Hf lung tumor reacted against cultured lung tumor cells in the microcytotoxicity assay. Reactivity was also observed against cells cultured from normal lungs of C3H and (C3Hf x A) F₁ mice but not against cells cultured from normal lungs of C3Hf or C57BL/6 mice. These results were interpreted to indicate that transplacentally induced malignant lung tumors of C3Hf mice express an antigenic component which exists as a normal tissue alloantigen, present in A and C3H but not in C3Hf, DBA/2 or C57BL/6 mice. It is suggested that the normal expression of the alloantigen in A mice may contribute to the susceptibility of these mice to the spontaneous development of lung tumors. The observation that a tumor bearing host has lymphoid cells reactive in the microcytotoxicity assay against tumor cells does not necessarily indicate that the tumor possesses a tumor specific antigen for in genetically susceptible tumor bearing hosts, the reaction may be directed against a self-antigen.

- 4474 EFFECT OF POLYINOSINIC-POLYCYTIDYLIC ACID ON HUMORAL AND CELLULAR IMMUNITY. (It.) Collavo, D. (Inst. Path. Anat., U. Padua, Italy), G. Biasi, N. Pennelli and L. Chieco-Bianchi. *Tumori* 59(2):77-96, 1973.

Polyinosinic-polycytidylic acid (Poly I:C), (0.1 mg i.p.), administered to RFM/Un mice 48 and 24 hr before 4 X 10⁸ sheep RBC, produced a marked reduction in direct plaque forming cells (PFC) and in the hemagglutinin titer on the 3rd, 4th and 5th days after antigen inoculation. However, an increase in PFC and hemagglutinin titer was observed on the 7th and 8th days. Histological examination revealed absence of germinal centers in the spleen on the 4th day. Poly I:C, administered 24 and 48 hr after antigen increased direct PFC and hemagglutinin titer on the 4th, 5th and 6th day. Germinal centers were found in the spleen on the 4th day after antigen. Poly I:C, administered 5 to 1 days before antigen, produced a markedly depressed direct PFC response in the groups injected 1 and 2 days before antigen. Recovery of the immune response was progressive and complete in groups injected 4 days before antigen. To study the effect of Poly I:C on secondary response to SRBC, two groups of animals injected with Poly I:C before or after antigen were reinjected with 2 X 10⁸ sheep RBC. Secondary response evaluated by hemagglutinin titer

at different intervals after immunization disclosed in both groups a much higher antibody titer than that in controls given only sheep RBC. Mice injected with Poly I:C 48 or 24 hr before reimmunization with 2 X 10⁸ sheep RBC did not differ from controls on 3rd and 4th days in regard to number of indirect PFC or hemagglutinin titer. Finally, mice immunized with two sheep RBC injections and then treated with Poly I:C on alternate days for 30 days had a much higher titer of hemagglutinins than controls. To study the effect of Poly I:C on the cellular immunity, spleen cells from animals receiving Poly I:C 6-5 days before sacrifice were cultured *in vitro* with phytohemagglutinin (PHA). DNA synthesis subsequent to PHA stimulation was evaluated by increase in ³H-thymidine incorporation. Cells from animals which had received Poly I:C had a much higher ³H-thymidine uptake than cells from control animals. Spleen cells obtained from RFM/Un mice injected with Poly I:C were inoculated in 1-4-day-old (RFM/Un x CBA/H)F₁ hybrids. Hybrids were then sacrificed on day 8 and spleen indices calculated. Experimental animals had graft *versus* host activity similar to that of controls (spleen index 2.3). Thus, if Poly I:C is injected before antigen the primary immune response is depressed, whereas it is enhanced when Poly I:C is administered after antigen. The secondary response is generally enhanced regardless of the time of Poly I:C administration. Moreover, in Poly I:C-treated animals there is an enhancement of PHA - responsive cells while the graft *versus* host reaction is unchanged. As Poly I:C is capable of enhancing immune reactivity, the possibility of its use in cancer chemotherapy is suggested.

- 4475 THE VARIABLE INFLUENCE OF HOST MICROFLORA AND INTERCURRENT INFECTIONS ON IMMUNOLOGICAL COMPETENCE AND CARCINOGENESIS. (E.) Hanna, M. G., Jr. (Carcinogenesis Program, Oak Ridge Natl. Lab., Tenn.), P. Nettesheim, C. B. Richter and R. W. Tennant. *Isr J Med Sci* 9(3):229-238, 1973.

Experiences with the influence of microbial environment on mammalian research, gathered over many years in the Biology Division of the Oak Ridge National Laboratory, are considered in a discursive manner and are examined in the light of current, pertinent data from other laboratories. The variable health status of animals used in research often affects the experimental results. Immunology and carcinogenesis are areas of experimentation especially vulnerable to the influence of "normal" microflora and intercurrent infections. Beyond the obvious effects of tissue pathogenetic changes, indigenous organisms and intercurrent infection are likely to influence various aspects of the immune response by competing with test antigen(s). The influence of intercurrent disease and indigenous viruses is especially marked in the events of carcinogenesis. The responses of mice to chemical and viral carcinogens may be affected by changes in the number of appropriate target cells, by alterations in immunologic capacity, by different cell turnover rates and by other factors related to the activation or deposition of the carcinogen and which can be influenced by indigenous or extrinsic

pathogens. The studies reviewed indicate potential areas of major variability associated with the microbial status of the host in both short- and long-term mammalian studies and underscore the need for experimental animals which are standardized for both autochthonous microflora and freedom from disease, as well as for genetic makeup.

- 4476 CELLULAR IMMUNITY TO BREAST CARCINOMA AND MALIGNANT MELANOMA. (E.) Cochran, A. J. (Western Infirmary, Glasgow, Scotland), R. M. Mackie, C. E. Thomas, R. M. Grant, D. E. Cameron-Mowat and W. G. S. Spilg. *Br J Cancer* 28(Suppl 1):77-82, 1973.

The effect of extracts of benign and malignant tumors on the migration of leucocytes from the peripheral blood of 55 melanoma patients, 73 breast carcinoma patients and 162 control donors have been examined in the leucocyte migration test. The leucocytes from a majority of tumor bearing patients were inhibited on contact with extracts of histogenetically similar tumors but only occasionally by extracts of dissimilar tumors and preparations from non-neoplastic breast tissue. Control donor's leucocytes were rarely inhibited by any of the tissue extracts. In both patients with malignant melanoma and those with breast carcinoma there was a striking decline in the frequency of tumor extract mediated leucocyte migration inhibition in patients with metastases. The authors have also observed when following patients serially, a transient post-operative loss of tumor extract induced migration inhibition, a phenomenon which may well be relevant to the establishment of metastatic deposits.

- 4477 PLASMA-CELL AND TUMOR-ASSOCIATED MEMBRANE ANTIGENS OF MOUSE PLASMACYTOMA MOPC-315 AND MOPC-460. (E.) Comoglio, P. M. (Turin U. Sch. Med., Italy) and G. Forni. *Int J Cancer* 12(3):613-625, 1973.

An immunological procedure was used to identify cell antigens solubilized from transplantable BALB/c MOPC-315 and MOPC-460 plasmacytomas. Out of the antigens solubilized from the mineral-oil induced plasmacytoma membranes, rabbit antisera and syngeneic mouse antisera recognized neoplastic plasma cell antigens that were not detectable in normal cells. The rabbit antisera showed precipitating antibodies against two different antigens expressed in both MOPC-460 and MOPC-315 cells and unrelated to plasma-cell specific antigens or myeloma proteins. The first antigen, embryo-antigen (EM-antigen), was also expressed by untransformed mouse embryo cells and seemed to be related to mitotic activity. The second, designated the A-particle-associated antigen (Aa-antigen), was shown by immuno-electron microscopy to be present on the virus-like A particle surface; it consisted of two related components and was found in all of nine different mouse tumors. In addition, one of the mouse antiserum pools contained precipitating antibodies against a third antigen of unique specificity expressed on MOPC-315 cells only; it was designated 315-tumor-specific antigen (315-TSA).

- 4478 MAMMARY TUMOR VIRUS (MTV) ANTIGENS IN NORMAL AND MAMMARY TUMOR-BEARING MICE. (E.) Hilgers, J. H. M. (Dept. Biol., Netherlands Cancer Inst., Amsterdam), G. J. Theuns and R. V. van Nie. *Int J Cancer* 12(3):568-576, 1973.

Quantitative immunofluorescence tests were used to study MTV-antigen production in normal and mammary tumor-bearing mice of two low (BALB/c and C3Hf) and two high (C3H and GR) mammary cancer strains. Mammary tumors could be distinguished on the basis of MTV-antigen expression in two groups: (1) a group of positive and (2) a group of negative tumors occurring in BALB/c mice. While the tumors of the first group were all adenocarcinomas, the tumors of the second group were adenoacanthomas. MTV-antigen production was comparatively high in tumors of the C3Hf and C3H strains, as well as in the MTV-induced tumors of the BALB/c strain, but low in GR-strain tumors. Sera from mice bearing MTV-antigen producing mammary tumors (as well as from some pregnant and lactating GR mice) contained excess MTV-antigens. The spleens of BALB/c mice bearing MTV-antigen-induced tumors often contained detectable amounts of MTV-antigens, while normal and mammary tumor-bearing C3Hf and C3H mice also occasionally showed detectable amounts of MTV-antigens in their spleens. In contrast the spleens of GR mice were always MTV-antigen negative by immunofluorescence absorption tests. The thymuses, livers and kidneys of the various strains tested were invariably negative.

- 4479 SIGNIFICANCE OF SERUM FACTORS MODIFYING CELLULAR IMMUNE RESPONSE TO GROWING TUMOURS. (E.) Baldwin, R. W. (Cancer Res. Campaign Lab., U. Nottingham, England), M. R. Price and R. A. Robbins. *Br J Cancer* 28(Suppl 1): 37-47, 1973.

Lymphocytes from hepatoma bearing rats are cytotoxic for cells of the corresponding hepatoma and this reactivity can be specifically blocked by pre-treating target cells with tumor bearer serum. This blocking activity is unlikely to be mediated by circulating tumor specific antibody, since none is detectable by cytotoxic assay in these sera. In contrast, serum from tumor immune rats is cytotoxic for plated hepatoma cells, but in the absence of complement these sera block lymphocyte cytotoxicity, findings which suggest that blocking by humoral antibody may not be a significant factor in modifying tumor growth. It has also been established that immune complexes prepared by adding hepatoma immune serum to solubilized tumor specific antigen can block target hepatoma cells from lymphocyte attack and this is probably the mechanism of blocking by tumor bearer serum. More significantly, lymph node cell cytotoxicity is specifically inhibited following incubation with tumor bearer serum. Similar lymphocyte inhibition is obtained by pretreating effector cells with solubilized hepatoma specific antigen. This suggests that the activity of tumor bearer serum may be effected by circulating tumor antigen or immune complexes and in support of this, there is evidence for these factors in the sera

studied. These investigations indicate that inhibition of lymphocyte reactivity by tumor bearer serum is of a complex nature, but probably the most relevant with regard to *in vivo* immune responses is the tumor-antigen mediated inhibition of lymphocyte reactivity.

4480 PRESENCE OF ANTIGEN COMMON TO AVIAN TUMOR VIRAL ENVELOPE ANTIGEN IN NORMAL CHICK EMBRYO CELLS. (E.) Hanafusa, H. (Public Hlth. Res. Inst. City New York, Inc., N.Y.), T. Aoki, S. Kawai, T. Miyamoto and R. E. Wilsnack. *Virology* 56(1):22-32, 1973.

Antiserum against the envelope antigen of avian tumor virus of subgroup E was obtained from chickens bearing tumors induced by the Bryan strain of Rous sarcoma virus produced from chf-positive C/E cells (RSV[f]). The capacity to absorb neutralizing activity from the antiserum was used to demonstrate the presence or absence of the envelope antigen in various types of cells. The specificity of the antigen was also confirmed by immuno-electron microscopy. Cell extracts or viable cells from the uninfected chick embryos were generally positive for the envelope antigen when the embryos were also positive in both the formation of gs-antigen and helper activity. Chick embryo cells negative for these viral functions were also deficient in the envelope antigen. However, two exceptional embryos were found in which the amount of envelope antigen and the level of helper activity do not directly correlate with each other. The antigen common to that on the viral envelope appeared to be located on or in the fuzzy coat at the cell surface.

4481 METHODS OF SOLID TUMOUR EVALUATION IN IMMUNOLOGICAL EXPERIMENT. I. OBJECTIVE ERROR IN THE DETERMINATION OF THE GROWTH CURVE. (E.) Rejthar, A. (Fac. Med., J.E. Purkyne U., Brno, Czechoslovakia), J. Wotke and P. Kellner. *Neoplasma* 0(3):271-279, 1973.

The importance of histological examination of tumors for accurate evaluation of their growth is demonstrated. A transplantable mammary carcinoma O-IV was grown in syngeneic CBA and resistant allogeneic C57B1 mice, using a challenge dose of cells sufficient to ensure growth in the resistant strain. Differences in tumor growth obtained with the two different strains of test animals were highly significant when evaluated by survival time of the animals or by rate of mortality due to the tumor. However, no significant differences were found when tumor growth was measured by rate of increase in mean tumor area. Histological examination of the tumors in syngeneic animals revealed invasive solid tumor tissue, hemorrhagic necrosis, and thin membranous noncollagenous tissue containing viable tumor cells which were capable of regenerating solid tumor. Examination of tumors in allogeneic animals showed mainly necrobiotic and granulation tissues; further, these tumors lacked the regenerative membranous tissue.

4482 CELL-MEDIATED CYTOTOXICITY AGAINST HeLa CELLS IN PATIENTS WITH INVASIVE OR PREINVASIVE CERVICAL CANCER. (E.) Saksela, E. (Dept. Pathol., U. Helsinki, Finland) and B. Meyer. *J Natl Cancer Inst* 51(4):1095-1102, 1973.

The feasibility of using HeLa cells, a continuous cell line from human cervical carcinoma, as targets in *in vitro* microcytotoxicity assays for cell-mediated immunity in patients with invasive or preinvasive carcinoma of the uterine cervix was investigated. The purified lymphocytes of 16 of 24 (67%) patients with invasive carcinoma and 14 of 27 (52%) with carcinoma *in situ* or severe epithelial dysplasia were cytotoxic against HeLa cells but not against target cells from an established line of human amnion grown for a comparable period *in vitro*. No specific HeLa cell cytotoxicity was found in patients with carcinoma of the uterine corpus or epidermoid carcinoma of the larynx nor among healthy controls. Sera could block the cell-mediated cytotoxicity in 50% of the 15 patients tested; this blocking occurred equally in both the preinvasive and invasive carcinomas. The results suggest that the HeLa cell line still carries tumor-specific surface antigens, which might be applied to the immunodiagnosis of human cervical carcinoma.

4483 Θ ALLOANTIGEN ON MOUSE AND RAT FIBROBLASTS. (E.) Stern, P. L. (Zool. Dept., U. Coll., London, England). *Nature [New Biol]* 246(151):76-78, 1973.

The expression of Θ alloantigens on fibroblasts and fibroblast-derived cell lines from mice and rats was studied using specific anti- Θ AKR antiserum prepared in CBA mice and anti- Θ C3H prepared in AKR mice. The presence of Θ on the cell surface was demonstrated by its ability to absorb the cytotoxic activity of the anti- Θ sera tested against the appropriated thymocytes. Fibroblasts and some of the fibroblast-derived cell lines were positive, carrying the allele appropriate to the strain of animal from which they were derived. The L cell and normal 3T3 cell lines tested, however, did not express the Θ C3H antigen. Mouse embryo fibroblasts contained about 10 times more/cell than CBA thymocytes, whereas polyoma-virus transformed 3T3 cells contained 10 times less. A normal mouse liver cell line, a methylcholanthrene-induced rat sarcoma derived from AKR positive rats, and a rat glial cell line derived from AKR negative rats all were negative. Thus, when testing for the presence of Θ on s.c. tumor cells, contribution for contaminating fibroblasts must be excluded.

4484 CHARACTERIZATION OF MEMBRANE ANTIGENS FROM ZAJDELA HEPATOMA AND NORMAL RAT LIVER. (Ger.) Rella, W. (Inst. Cancer Res., Univ. Vienna, Austria) and B. Chaput. *Arch Geschwulstforsch* 41(4):301-310, 1973.

A modification of Neville's method was used to fractionate cells of Zajdela hepatoma, passaged in the

ascites form in adult Sprague-Dawley rats, and of liver from normal Sprague-Dawley rats into cytoplasmic membrane and endoplasmic reticulum fractions. Comparisons were made of enzyme activities, effects on the cytotoxic activity of rabbit antihepatoma sera, and the effect of ultrasound on the antigenic activities of these fractions. In contrast to normal rat liver, Zajdela hepatoma cells did not contain 5'-nucleotidase, which is specific for cytoplasmic membranes, or glucose-6-phosphatase, which is specific for microsomes. With the exception of one fraction of normal liver cells, only hepatoma cell fractions were able to inhibit the cytotoxic activity of rabbit antihepatoma sera. In contrast to other findings in the literature, antigenic activity was found primarily in the endoplasmic reticulum. The antigenic activity of the one fraction of normal liver cells may either represent a true cross reaction between normal and tumor tissue, or it may result from antilymphocyte components in the antisera since the ascites hepatoma cells were contaminated with 2-5% lymphocytes. When exposed to ultrasound (70 watts for one 20 sec period or two 60 sec periods), antigenic activity of cytoplasmic membrane fractions from tumor cells doubled while that of endoplasmic reticulum fractions remained essentially unchanged. Part of this increase in antigenic activity may be due to the breaking down of these larger particles into smaller ones. However, antigenic activity of endoplasmic reticulum fractions was still greater than that of cytoplasmic membrane particles, and this difference could not be explained solely by differences in the sizes of particles in these fractions.

- 4485 CROSS-REACTIVITY BETWEEN CELL-SURFACE ANTIGENS OF DIFFERENT MURINE CARCINOGEN-INDUCED TUMORS, DEMONSTRATED BY A MODIFIED ISOTOPIC ANTIGLOBULIN TEST. (E.) Burdick, J. F. (Natl. Cancer Inst., Bethesda, Md.) and S. A. Wells, Jr. *J Natl Cancer Inst* 51(4):1149-1156, 1973.

The isotopic antiglobulin test modified for use with tissue culture cells grown as adherent monolayers resulted in a quick, sensitive microassay. Serum, prepared from blood of mice hyperimmunized with a syngeneic C57BL/6N tumor induced by 3-methylcholanthrene (MCA) *in vitro*, cross-reacted with three of seven different MCA-induced tumors. After absorption of serum, the activity was blocked by all MCA-induced tumor lines and a normal fibroblast line, but not by normal spleen cells. There was a weak cross-reactivity with murine Moloney and Gross virus-induced tumors. A similar weak reactivity with C57BL/6N multiparous exbreeder serum indicated a possible relationship to fetal antigen.

- 4486 IMMUNOLOGIC STATUS IN LUNG CANCER. (E.) Brugarolas, A. (Dept. Thoracic Surg., Roswell Park Mem. Inst., Buffalo, N.Y.) and H. Takita. *Chest* 64(4):427-430, 1973.

Delayed cutaneous hypersensitivity was studied in 219 patients (176 males and 43 females, aged 35-83 yr) with carcinoma of the lung to observe if there

was a correlation between the immunologic status of the patients and the major clinical features of the disease. Delayed cutaneous hypersensitivity correlated with the cell type differentiation of the tumor, the stage of the disease, the response to chemotherapy, the prognosis and the survival. Patients with anergia often had undifferentiated cell tumors (anaplastic, oat cell, and giant cell type), failed to respond to therapy, had advanced disease and died in a short time. Patients with a good skin test reaction tended to have well-differentiated carcinomas (squamous and glandular), often had limited disease, responded more frequently to therapy and survived for longer periods. In the 21 patients studied, a good correlation was found between skin test reactions and *in vitro* lymphocyte transformation.

- 4487 SURFACE ANTIGENS COMMON TO MOUSE CLEAVAGE EMBRYOS AND PRIMITIVE TERATOCARCINOMA CELLS IN CULTURE. (E.) Artzt, K. (Inst. Pasteur, Paris, France), P. Dubois, D. Bennett, H. Condamine, C. Babinet and F. Jacob. *Proc Natl Acad Sci USA* 70(10):2988-2992, 1973.

Syngeneic antisera were produced in mouse strain 129/Sv-CP males against the primitive cells of teratocarcinoma. These sera react specifically with the primitive cells and are negative on various types of differentiated teratoma cells derived from the same original tumor. They are negative on all other mouse cells tested, with the exception of male germ cells and cleavage-stage embryos. Thus, teratoma cells possess cell-surface antigens in common with normal cleavage-stage embryos. The number of antigens observed is unknown. Primitive teratoma cells are highly tumorigenic, and some tumor antigens might be present on their cell surfaces. No cross reaction was observed however, with various viral tumor antigens. The most significant result reported is the reaction of antisera against primitive teratoma cells with mouse morula cells. The reaction appears to increase from very little, if any, with 1-cell eggs to a maximum at the 8-cell stage. This result suggests the progressive expression of a new surface antigen(s) during the earliest stages of development.

- 4488 PREPARATION OF MEMBRANE FRACTIONS WITH ENHANCED TUMOR-TRANSPLANTATION-ANTIGEN ACTIVITY FROM TUMOR CELLS INFECTED WITH INFLUENZA VIRUS. (E.) Boone, C. W. (Natl. Cancer Inst., Bethesda, Md.), T. W. Orme, K. Blackman and R. Gillette. *J Natl Cancer Inst* 51(4):1141-1144, 1973.

Cell membrane fragments isolated from homogenates of influenza virus-infected E4 tumor cells, derived from a fibrosarcoma in a BALB/c mouse inoculated with SV40-transformed BALB/3T3 cells, are shown to possess tumor transplantation antigen (TTA) activity approaching that of the intact X-irradiated tumor cells. This enhancement of immunogenicity was specific: Immunization of mice with homogenates of influenza virus-infected normal cells or spontaneously transformed tumor cells did not confer immunity

to challenge with the SV40-tumor cells. The virus-enhanced TTA activity was associated with cell membrane fractions isolated either on sucrose density gradients or from phosphate-buffered saline eluates of frozen and thawed tumor cells.

- 4489 AUGMENTATION OF THE ADOPTIVE TRANSFER OF SPECIFIC TUMOR IMMUNITY BY NONSPECIFICALLY IMMUNIZED MACROPHAGES. (E.) Ariyan, S. (Yale U. Sch. Med., New Haven, Conn.) and R. K. Gershon. *J Natl Cancer Inst* 51(4):1145-1148, 1973.

The ability of normal and BCG-immune peritoneal exudate cells to assist tumor-immune lymph node cells in tumor killing was studied. The adoptive transfer of 9×10^6 tumor-immune lymph node cells mixed with 3×10^6 BCG-immune peritoneal exudate cells to normal syngeneic hamsters significantly suppressed the growth of 3×10^6 tumor cells. Neither cell population alone or combined with nonimmunized cells affected tumor growth at these cell concentrations. Thus nonspecifically immunized macrophages could partake in tumor killing *in vivo*, but to do so they required the assistance of specifically immunized cells, most likely lymphocytes. This form of cooperative interaction between lymphoid cells and macrophages differed somewhat from previous reports in that the addition of the antigen to which the macrophages were sensitized was not required for the cooperative event to occur. It is suggested that, in the presence of specific tumor antigen, immune lymphocytes can cause macrophages to release their contents that are toxic for some "bystander" cells. However, some tumor cells are resistant to the products released by normal macrophages but may be sensitive to those from nonspecifically immunized or "activated" macrophages.

- 4490 SUGGESTED CORRELATION BETWEEN RADIATION-INDUCED IMMUNOSUPPRESSION AND RADIOGENIC LEUKEMIA IN MICE. (E.) Clapp, N. K. (Biol. Division, Oak Ridge Natl. Lab., Tenn.) and J. M. Yuhas. *J Natl Cancer Inst* 51(4):1211-1215, 1973.

The reduced incidence of reticulum cell sarcoma in RF mice with glomerulosclerosis (GLO) and the demonstration that GLO partly results from an immune response to wild-type virus-associated antigens have provided a means for the determination of the importance of radiation-induced immunosuppression in leukemogenesis. This finding of reduced leukemia risk in mice with GLO has now been extended to include the two radiation-induced types, thymic and myeloid. The incidence of radiation-induced leukemia increased with radiation dose in both GLO-positive and GLO-negative mice, but the presence of GLO was associated within each dose group with a twofold reduction in risk for each leukemia. Doses >100 rads suppressed the immune system and the development of GLO and thereby shifted a fraction of the animals from the low-risk (GLO-positive) group to the high-risk (GLO-negative) group. Since GLO is, at least in part in these RF mice, the product of an immune reaction against a virus-associated antigen, suppressing the

ability to cope with leukemogenic virus-associated antigens (suppressing the immune system) contributed to the overall leukemogenic effectiveness of radiation, at least for doses >100 rads. Since suppression of GLO development did not contribute in the lower dose range (<100 rads) clearly the dose-response curve for radiation-induced leukemia in the RF mouse cannot be linear.

- 4491 CIRCULATING ANTIGEN-ANTIBODY COMPLEX ASSOCIATED WITH EPSTEIN-BARR VIRUS IN RECURRENT BURKITT'S LYMPHOMA. (E.) Mukojima, T. (Natl Cancer Ctr. Hosp., Tokyo, Japan), P. Gunven and G. Klein. *J Natl Cancer Inst* 51(4):1319-1321, 1973.

Four yr after initial diagnosis and treatment, the sera from a patient with recurrent Burkitt's lymphoma showed a sudden decrease of antibodies to Epstein-Barr virus (EBV)-associated membrane antigens (MA). This decrease occurred several months before the recurrent tumor became clinically apparent. Simultaneously, the sera developed a high anticomplementary activity (ACA). The ACA was associated with the globulin fraction as demonstrated by ammonium sulfate precipitation. Low pH treatment of the globulin of one serum and Amicon filtration restored anti-MA reactivity to the fraction retained above the filter (molecules $>10^5$ Daltons). These data seem to indicate that MA-antibody complexes were present in the patient's serum. If EBV is a necessary factor in the pathogenesis of BL, and/or if antibodies against MA are significant in restricting the growth of dormant BL clones, these events could conceivably promote recurrence.

- 4492 LYMPHOCYTE POPULATIONS OF AKR/J MICE. I. EFFECT OF AGING ON MIGRATION PATTERNS, RESPONSE TO PHA AND EXPRESSION OF THETA ANTIGEN. (E.) Zatz, M. M. (Albert Einstein Coll. Med., Bronx, N.Y.), A. L. Goldstein and A. While. *J Immunol* 111(5):1514-1518, 1973.

Evidence is presented that there is a distinct change in the lymphoid populations in lymph node, spleen, and thymus of aging AKR/J mice. These mice have a high incidence of spontaneous lymphocytic leukemia and lymphoma beginning at about 6 months of age and they carry Gross leukemia virus from birth. Three parameters were chosen to study lymphoid populations: 1) *in vivo* migration of ^{51}Cr labeled lymphocytes to normal recipient lymph nodes, 2) incorporation of ^3H -thymidine by lymphocytes *in vitro* in response to phytohemagglutinin (PHA), and 3) expression of the theta (θ) AKR antigen on lymphoid cells *in vitro*. These three characteristics of lymphocytes have all been shown to be manifested by thymus-dependent (T) cells and were therefore used to quantitate changes in T cell populations of AKR/J mice as a function of age. Calculated numbers of lymph node-seeking cells (LNSC) in lymph node, spleen, and thymus of AKR/J mice aged 3 to 14 months show a marked decline in the size of the lymph node-seeking component of all three compartments with age. No significant change is apparent in the representation of θ antigen on AKR/J lymph node cells with age.

Stimulation of DNA synthesis by addition of PHA to spleen cell cultures from 3-, 6-, 10-, and 14-month old AKR/J mice revealed a clear decline in the stimulation ratio of spleen cells from mice aged 3 to 14 months. The decline in the T-cell population of AKR/J spleen correlates temporally to the onset of leukemogenesis at about 6 months of age. At least two possible mechanisms may be suggested as a basis for the coincidental decrease in the T population and the appearance of leukemia in the AKR/J strain: 1) a change in lymphoid populations preliminary to leukemogenesis, and 2) leukemogenesis preliminary to changes in the lymphoid populations. Based on previous studies in leukemic mice and from age studies in other strains it is believed that the decline in the T population of AKR/J mice occurs preliminary to, and possibly predisposing to, onset of leukemia linked to the endogenous presence of the Gross murine leukemia virus in this strain.

- 4493 DISTURBANCE OF CELL-MEDIATED IMMUNITY IN PATIENTS WITH CARCINOMA OF COLON AND RECTUM. (E.) Manousos, O. N. (Second Dept. Med., Athens U., Greece), J. Economidou, Ch. Pathouli and G. Merikas. *Gut* 14(9):739-742, 1973.

The cellular immune mechanisms of patients (14 male and 6 female, ranging in age from 33 to 75 yr) with adenocarcinoma of colon or rectum were evaluated by studying the lymphocyte response to phytohemagglutinin stimulation. The results indicate that the serum of cancer patients causes a decreased blastic transformation and H^3 thymidine uptake of the lymphocytes of patients as well as of normal controls. A defect of the lymphocytes unrelated to serum factors was also present in some cancer patients. However, no relationship was found between the degree of transformation and the presence of metastases in the lymph nodes. In certain (or perhaps all) cancer patients it appears that there is an interplay between cellular reactions mediated by lymphocytes and serum factors (enhancing antibody) antagonizing the cell reaction of the host.

- 4494 LYMPHOCYTE POPULATIONS OF AKR/J MICE. II. EFFECT OF LEUKEMOGENESIS ON MIGRATION PATTERNS, RESPONSE TO PHA, AND EXPRESSION OF THETA ANTIGEN. (E.) Zatz, M. M. (Albert Einstein Coll. Med., Bronx, N.Y.), A. White and A. L. Goldstein. *J Immunol* 111(5):1519-1525, 1973.

The present study describes abnormalities that occur in the lymph node, spleen, thymus, and blood lymphocytes of two distinct types of leukemic AKR/J mice. These two types, designated A and B leukemia, occur in a ratio of 3:1 in mice aged 7 to 10 months. Type A leukemic mice are characterized by the presence of a thymoma and enlarged peripheral lymphoid organs, and lymphocytes which 1) fail to migrate to lymph nodes, 2) exhibit normal transformation in response to PHA, and 3) have elevated sensitivity to anti-theta (θ) serum. Type B leukemic mice have an atrophic thymus and enlarged peripheral lymphoid organs; their lymphocytes 1) fail to home to lymph nodes, 2) exhibit decreased transformation

in response to PHA, and 3) have decreased sensitivity to anti- θ serum. These findings are compatible with the possibility that type A leukemia is thymus-dependent and preventable by thymectomy, whereas type B leukemia is less thymus dependent and may occur even after removal of the thymus. Preliminary histologic findings (unpublished observations) show type A leukemia to involve thymus dependent areas of lymphoid tissues, and type B leukemia to be of a more anaplastic, and less readily categorized form. The aberrations observed in lymphocytes of AKR/J leukemic mice may reflect a complex of cell surface changes, affecting distinct molecules involved in the regulation of lymphocyte recirculation, binding of PHA, and expression of θ . It is possible that the changes in AKR/J lymphocyte populations observed during leukemogenesis reflect depletion of certain subsets of lymphocytes and proliferation and expansion of others, and the origins of type A and B leukemia are distinct.

- 4495 *IN VITRO* INDUCTION OF TUMOR SPECIFIC IMMUNITY: REQUIREMENTS FOR T LYMPHOCYTES AND TUMOR GROWTH INHIBITION *IN VIVO*. (E.) Röllinghoff, M. (Walter and Eliza Hall Inst. Med. Res., Melbourne, Australia) and H. Wagner. *Eur J Immunol* 3(8):471-476, 1973.

Cortisone-resistant thymocytes, spleen cells, thoracic duct lymphocytes, peritoneal exudate cells and peripheral blood lymphocytes of BALB/c mice were immunized *in vitro* against syngeneic HPC-108 plasma cell tumor cells. Cocultivation of spleen lymphocytes together with HPC-108 cells generated the highest cytotoxic immune response in comparison to other lymphocyte sources. Cytotoxicity was tested in a 6 hr ^{51}Cr release assay using HPC-108 cells as target cells. The use of AKR anti- θ C3H serum indicated that thymus-derived (T) lymphocytes are essential to the initiation phase of the immune response to plasma cell tumor cells. Furthermore, evidence is presented that the cytotoxic effector cells in the *in vitro* tumor immune response are T lymphocytes. Spleen cells activated *in vitro* against HPC-108 tumor cells were shown to specifically prevent tumor growth from simultaneously injected HPC-108 cells in irradiated recipient mice.

- 4496 HEPATOMA GROWTH. ENHANCEMENT AFTER IMMUNIZATION WITH PLASMA MEMBRANES AND ADJUVANT. (E.) Orlando, R. A. (California Coll. Med., Irvine), K. Craft, M. Glick, C. Barbour and R. W. Wissler. *Arch Pathol* 95(4):229-234, 1973.

Plasma membranes isolated from a Morris hepatoma No. 5123 were evaluated as a source of effective tumor-specific antigens. They were combined with methylated bovine serum albumin and pertussis vaccine and injected into syngeneic female Buffalo rats. The injections were repeated 10 days later, and 4 days after the second injection, the animals were inoculated with viable hepatoma cells. Eleven days later, palpable subcutaneous tumor nodules were observed in 93.5% of the animals immunized

with the hepatoma membranes; similar tumors were present in 73.5% of the animals immunized with whole hepatoma cells and 20% to 33.3% of the other control animals (immunized with normal liver, normal liver membranes, hepatoma cells combined with bovine serum albumin and pertussis, or saline). In addition, the mean tumor volume of the animals immunized with the hepatoma membranes was 107 cc, as compared with 85.3 cc for the whole-cell immunized rats and 53.7 to 65.1 cc for the control rats. In a second experiment, tumors appeared in 91.5% of the rats immunized with hepatoma membranes, 83.5% of those immunized with whole hepatoma cells combined with methylated bovine serum albumin and pertussis, and 8.4 to 33.3% of the control animals. Thus, the combination of plasma membranes from a hepatoma with pertussis vaccine and methylated bovine serum albumin produces an effective, tumor-specific, antigenic complex capable of stimulating enhancement in syngeneic Buffalo rats.

4497 SURFACE MARKERS ON HUMAN B AND T LYMPHOCYTES. II. PRESENCE OF EPSTEIN-BARR VIRUS RECEPTORS ON B LYMPHOCYTES. (E.) Jondal, M. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden) and G. Klein. *J Exp Med* 138(6):1365-1378, 1973.

Epstein-Barr virus (EBV)-producing cells of the P3HR-1 cell line were mixed with human peripheral lymphocytes. Unfractionated lymphocytes were found to adhere to these cells, which displayed fresh viral determinants in their cytoplasmic membrane, in contrast to column-purified T lymphocytes. The specificity of the binding was confirmed by blocking experiments that showed that sera containing high titers of antibodies directed against the virus could partially inhibit the adherence in contrast to low-titer sera. It is concluded that B lymphocytes, in contrast to T lymphocytes, have receptors for EBV. In a second line of experiments, it was found that established human lymphoblastoid lines that carry the EBV genome had receptors characteristic for B lymphocytes and did not form T-lymphocyte rosettes. In contrast, a line of known T-lymphocyte origin that did not carry the EBV genome had receptors characteristic for T lymphocytes. EBV-transformed simian lymphoblastoid lines had surface markers indicating a B-lymphocyte origin in contrast to herpesvirus Saimiri-transformed simian lines that lacked surface immunoglobulin but carried receptors for sheep red blood cells. These data suggest that EBV acts on the bone marrow-derived lymphocyte system in man.

4498 THE HUMORAL ANTIBODY RESPONSE TO A PRIMARY VIRAL NEOPLASM (MSV) THROUGH ITS ENTIRE COURSE IN BALB/c MICE. (E.) Lamon, E. W. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), E. Klein, B. Andersson, E. M. Fenyo and H. M. Skurzak. *Int J Cancer* 12(3):637-645, 1973.

Forty adult BALB/c mice injected with Moloney sarcoma virus (MSV) developed, within 5-12 days, tumors at the site of inoculation which usually completely regressed by day 20-25. Maximal tumor size was

reached around day 15. Sera from these animals were examined for complement-dependent cytotoxic activity against Moloney lymphoma cells. The cytotoxic antibody response was biphasic. The first peak was reached at day 10 and extended to day 15. Twenty days after virus infection in the early regressor sera the antibody titers fell to normal serum values, followed by a progressive rise which persisted in the long-term regressors during the entire observation period of 3 to 6 months. Active sera from days 10, 15, 30 and long-term regressors were pooled separately and fractionated on Sephadex G-200 into 19S and 7S fractions, and reconstituted to the original volume. The fractions were then tested for cytotoxic activity. Both the 19S and 7S fractions of all pools contained cytotoxic activity. Immunofluorescence analysis of these fractions showed the 7S, but not the 19S, fractions to be detectable by membrane immunofluorescence. Virus neutralizing activity was found in both 19S and 7S fractions, with higher activity in the sera from regressor animals.

4499 IMMUNITY TO TUMOURS OF THE MURINE LEUKAEMIA-SARCOMA VIRUS COMPLEX. (E.) Owen, J. J. T. (I.C.R.F. Tumour Immunol. Unit, University Coll., London, England) and R. C. Seeger. *Br J Cancer* 28(Suppl. 1):26-39, 1973.

Using an isotopic (^{125}I UdR) adaptation of the Hellström colony inhibition assay, two mechanisms of growth inhibition of murine sarcoma virus-murine leukemia virus (Moloney) sarcoma cells by immune lymph node cells from BALB/c mice have been demonstrated *in vitro*. One mechanism is not specific for MSV-MLV targets since a significant inhibitory effect was found with polyoma-3T3 cells and primary BALB/c embryo fibroblast cultures as targets; this mechanism is probably mediated by macrophages. The other mechanism is specific for MSV-MLV targets and is mediated by nonadherent lymph node cells.

4500 ANTIBODY TO EPSTEIN-BARR VIRUS IN MAN IN AUSTRALIA AND NEW GUINEA. (E.) Pope, J. H. (Queensland Inst. Med. Res., Brisbane, Australia), M. K. Walters, W. Scott and F. W. Gunz. *Int J Cancer* 12(3):689-698, 1973.

Antibody to Epstein-Barr (EB) virus was assayed in sera from leukemic and control groups in Australia and New Guinea. The prevalence of antibody to EB virus in a normal population in Brisbane increased gradually from about 31% in 1- to 5-year-olds to about 90% in adults. A similar age distribution occurred in a population from Griffith, N.S.W. In contrast, all 1- to 5-year-old Aboriginal children from the west coast of Cape York Peninsula and from New Guinea had antibody to EB virus, indicating a high rate of infection in early life. Aborigines from the east coast of Cape York Peninsula had a slightly lower prevalence. The geometric mean (GM) titers of 63 leukemic cases from Sydney (1:72) and of 60 non-leukemic hospital patients (1:80) were not significantly different, but each was significantly

higher than that (1:44) of 57 normal controls from Griffith. No association was found between the degree of remission in 29 cases of acute myeloid leukemia and either the presence of antibody to heat-labile EB viral complement-fixing antigen or the titer of antibody to viral capsid antigen, or to crude or soluble EB viral complement-fixing antigens. The significantly higher GM antibody titers in non-leukemic patients and in Aboriginal children with non-malignant diseases suggest that some pathological process common to a variety of diseases results in moderately increased titers of antibody to EB virus.

- 4501 TUMOUR SPECIFIC CELL SURFACE ANTIGEN AND FORSSMAN ANTIGEN ASSOCIATED WITH RAT AND HAMSTER CELLS TRANSFORMED WITH DIFFERENT STRAINS OF AVIAN SARCOMA VIRUS. (E.) Michele, C. A. (Unit. Virol. I.N.S.E.R.M., Lyon, France), D. Simkovic and L. Gazzolo. *Neoplasma* 20(5):481-490, 1973.

Rat and hamster cells transformed by three different strains of avian sarcoma virus (SR-RSV, PR-RSV, and B77V) were found to contain at least two different surface antigens: a virus-induced surface antigen (S antigen), and Forssman antigen (F antigen). Both antigens were revealed on the surfaces of virus-producing, virogenic, and non-virogenic living cells by the method of mixed hemadsorption, using heterologous rabbit serum anti RS2/10 hamster tumor cells. The results of blocking tests, in which the cells were incubated with adsorbed rat anti RS2/10 serum and then with the rabbit anti Forssman serum, indicate that the S and F antigens are different. It is possible that a part of the ASV genome is integrated within the transformed cells, although it appears more likely that the induction of S and F antigens is similar to the phenomenon of conversion of somatic antigens in some strains of Salmonella.

- 4502 GROWTH AND MALIGNANT CONVERSION OF FOETAL TISSUES IN SYNGENEIC ECTOPIC TRANSPLANTATION. (E.) Zinzar, S. N. (Inst. Exp. Clin. Oncol., Acad. Med. Sci, Moscow, USSR), G. J. Svet-Moldavsky, B. G. Tumyan and B. I. Leitina. *Neoplasma* 20(5):531-538, 1973.

Embryonic digestive tract (DT) (as a whole, in parts, in pieces, or as minced tissue) was transferred to skin-allograft-bearing mice of the BALB/c, C57B1/6j, CBA, and DBA-2 strains. The DT tissues grew progressively in the hosts, forming cysts filled with sterile secretion. When transplanted subcapsularly into the spleens and kidneys of host mice, the DT formed cysts filled with mucosa. With time the structure of the cysts changed, some of them undergoing spontaneous malignant conversion: 13 to 16 months after grafting, tumors were observed in 6 out of 10 mice. In a second experiment, the growth of fetal intestine transplants in newborn and adult recipients was observed. The stomach and large intestine cysts appeared in 100% of the newborn mice, but appeared later than in the adult recipient. The

small intestine transplants in the newborn recipients not only appeared later than in the adults, they often did not appear at all. Further, the small intestine transplants grew better in newborn females than in newborn males. Thus, in the third experiment, the small intestines of males were transplanted into newborn males, while newborn females received transplants from females. Transplantation cysts developed in 20% of the males and 50% of the females. When tumors formed after the spontaneous malignant conversion of the cysts of different parts of the DT were transplanted, tumor nodes of leiomyosarcoma transplants appeared a week later in the newborn recipients than the adult recipients; however, the nodes reached greater weights in the newborns. With transplanted adenocarcinoma of large intestine, tumor nodes appeared at relatively the same time in some newborn and adult mice, although they were soon observed in all newborns, in which they attained greater weights. In contrast, tumor nodes of transplants of adenocarcinoma of small intestine appeared much later in newborns, not appearing in all newborns until 3 to 4 months later. It is suggested that in the embryonic period and early childhood, there are some factors specifically preventing the multiplication of cells of embryonic small intestine and tumors of small intestine.

- 4503 SURFACE ANTIGEN ON HAMSTER CELLS TRANSFORMED *IN VITRO* BY TWO STRAINS OF ROUS SARCOMA VIRUS. (E.) Bataillon, G. (Radium Inst., Sci. Fac., Orsay, France). *J Gen Virol* 21(1):175-179, 1973.

Hamster cells transformed *in vitro* by the Bryan (RB 12) and the Schmidt-Ruppin (RS 2) strains of Rous sarcoma virus (RSV) were studied for the presence, at their surface, of the virus-induced surface antigen (VISA). VISA expression, as detected by an *in vitro* colony inhibition test, was higher in the RS 2 cells than in the RB 12 cells. Contrary to other observations which suggest that there is a positive correlation between group-specific antigen content and VISA expression in hamster cells transformed by Schmidt-Ruppin RSV, the present results suggest that the two antigens are submitted to quite unrelated regulation controls when RB 12 and RS 2 cells are compared. Cytotoxic antisera directed against the two kinds of cells cross-reacted. Chicken cells infected by a non-transforming virus (RAV-1) did not elicit the appearance of detectable cytotoxin antibodies in hamsters.

- 4504 EFFECT OF REMOVAL OF BLOCKING ANTIBODIES BY IMMUNOADSORPTION ON THE GROWTH OF ASCITES TUMORS. (E.) Ambrus, J. L. (Roswell Park Mem. Inst., Buffalo, N.Y.) and C. M. Ambrus. *Proc Am Assoc Cancer Res* 14(March):119, 1973.

4505 STUDIES CONCERNING THE REGIONAL LYMPH NODE IN CANCER. III. RESPONSE OF REGIONAL LYMPH NODE CELLS FROM BREAST AND COLON CANCER PATIENTS TO PHA STIMULATION. (E.) Fisher, B. (U. Pittsburgh Sch. Med., Pa.), E. A. Saffer and E. R. Fisher. *Cancer* 30(5):1202-1215, 1972.

4506 IMMUNOLOGIC ASPECTS OF LEUKEMIA. (E.) Weiden, P. L. (United States Public Hlth. Service Hosp., Seattle, Wash.). *N Engl J Med* 288(16):852, 1973.

4507 T AND B LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKEMIA. (E.) Cabanillas, F. (U. Hosp., Rio Peidras, Puerto Rico). *N Engl J Med* 288(16):852, 1973.

4508 *IN VITRO* SYNTHESIS OF DNA COMPLEMENTARY TO mRNA DERIVED FROM A LIGHT CHAIN-PRODUCING MYELOMA TUMOUR. (E.) Aviv, H. (Natl. Inst. Hlth., Bethesda, Md.), S. Packman, D. Swan, J. Ross, P. Leder. *Nature [New Biol]* 241(110):174-176, 1973.

4509 HL-A AND MALIGNANT MELANOMA. (E.) Cordon, A. L. (Westminster Hosp, London, England). *Lancet* (7809):938, 1973.

4510 MACROPHAGE MIGRATION INHIBITION FACTOR FROM HUMAN LYMPHOCYTES IN CULTURE WITH NEOPLASTIC CELLS. (E.) Reid, R. H. (Madigan Gen. Hosp., Tacoma, Wash.). *Proc Am Assoc Cancer Res* 14(March):126, 1973.

4511 STRAIN SPECIFIC TA3K1 TUMOR CELLS IMMUNIZE ALLOGENEIC HOSTS AGAINST THE NON-SPECIFIC TA3Ha TUMOR. (E.) Lippman, M. (Natl. Inst. Hlth., Bethesda, Md.), J. Venditti, S. A. Schepartz, I. Kline and D. Elam. *Proc Am Assoc Cancer Res* 14(March):121, 1973.

See also:

- * (Rev): 4206, 4207, 4233, 4242, 4251, 4254, 4257
- * (Chem): 4283
- * (Viral): 4361, 4362, 4376, 4400, 4401, 4402, 4406, 4435, 4445, 4452, 4454

4512 ATYPICAL MEGAKARYOCYTES IN PRELEUKEMIC PHASE OF ACUTE MYELOID LEUKEMIA. (E.)

Smith, W. B. (Dept. Pediatrics, U. California, San Francisco), A. Ablin, J. R. Goodman and G. Brecher. *Blood* 42(4):535-540, 1973.

Mono- and binucleated megakaryocytes appeared in the bone marrow of a 6-yr-old white male 9 months prior to the development of acute myeloid leukemia; in addition, leukopenia and anemia antedated the diagnosis by 3 1/2 and 9 months resp. The report suggests that mono- and binucleated megakaryocytes may be a useful diagnostic finding in early leukemia. The megakaryocytes were very small with suggestion of coarse azurophilic inclusions. Electron microscopy revealed megakaryocytes containing one nucleus and, very rarely, in cross section, a second nuclear lobe was transected. The nuclear chromatin indicated a mature cell. Other abnormalities included areas with cytoplasmic blebs, many Golgi vesicles, and an apparent increase of small rounded mitochondria. Vesicular areas devoid of any other organelles were present in many megakaryocytes and resembled similar areas in megakaryocytes of hereditary macrothrombocytopathia.

4513 THE ESTHESIONEUROEPITHELIOMA. (E.)

Bottema, T. (Amsterdam, Netherlands). *Otorhinolaryngology Digest* 35(3):141-145, 1973.

Esthesioneuroepithelioma (olfactory neuroblastoma) is a rare tumor. In the 134 cases reported in the literature up to 1971, two types are distinguished: the esthesioneurocytoma consisting of cords or irregular islands of undifferentiated cells, separated by septa of connective tissue and sometimes with the formation of pseudorosettes and neurofibrils; and the esthesioneuroepithelioma with the formation of true rosettes. The clinical picture is that of a polypoid mass high in the nose, which bleeds easily and causes nasal obstruction. Rats injected s.c. with diethylnitrosamine in doses of 7.5 mg/kg twice weekly until the animals died survived an average time of 221.9 days (mean:174 - 245 days). All rats showed extensive tumors in the nose, and esthesioneuroepitheliomas in the olfactory region were observed. In a later similar experiment, a certain number of animals were killed every month in order to study the site of origin and growth of these tumors. These experiments and clinical experience have shown that the basal cell layer of the olfactory epithelium is the site of origin of the esthesioneuroepithelioma.

4514 OCCURRENCE AND PATHOGENESIS OF CHILDHOOD THYROID CANCER. (E.) Silink, F. (Res.

Inst. Endocrinology, Prague, Czechoslovakia), J. Soumar and J. Nemec. *Neoplasma* 20(4):465-469, 1973.

In the period 1953-1971, thyroid cancer was verified in 38 children under 15 yr of age in Czechoslovakia. The majority of cases occurred between 14-15 yr. Radiation-induced thyroid cancer is rare. Therapeutic irradiation to the thyroid region had been performed on only three patients. An additional three

patients had previously been subjected to repeated roentgenological examination. It is believed that some factors related to endemic goiter distribution may play a role in the genesis of the disease. The causative role of simple iodine deficiency is doubted. However, iodide prophylaxis may suppress the effect of thyroid stimulating hormone (TSH). TSH by itself may be an initiating factor or a promotive factor may be dependent on the TSH level.

4515 ON THE CLINICAL COURSE OF GASTRIC POLYPS.

(Rus.) Mirzaev, A. P. (Sanitary Hygiene Med. Inst., Leningrad, USSR) and I. V. Azarova. *Vopr Onkol* 19(8):26-29, 1973.

4516 C-CELL HYPERPLASIA PRECEDING MEDULLARY THYROID CARCINOMA. (E.) Wolfe, H. J.

(New England Med. Ctr. Hosp., Boston, Mass.), K. E. W. Melvin, S. J. Cervi-Skinner, A. A. Al Saadi, J. F. Juliar, C. E. Jackson and A. H. Tashjian, Jr. *New England J Med* 289(9):437-441, 1973.

4517 THE GENESIS OF GLANDLIKE CHANGES OF UTERINE CERVIX. (E.) Volfson, N. I. (USSR)

Ministry Public Hlth., Leningrad). *Neoplasma* 20(2):189-196, 1973.

4518 EXCRETION OF THE ADRENAL CORTEX HORMONES IN PRETUMOR PROCESSES AND CANCER OF THE UTERINE BODY. (Rus.) Bogdanova, A. G. (Kazakh Res.

Inst. Oncology, Radiology, Alma-Ata, U.S.S.R.) and N. N. Mezinova. *Vopr Onkol* 19(7):27-31, 1973.

4519 CHRONIC LYMPHOCYTIC LEUKAEMIA OF T-CELL ORIGIN? (E.) Sumiya, M. (Fac. Med., U.

Tokyo, Japan), H. Mizoguchi, K. Kosaka, Y. Miura, F. Takaku and J.-I. Yata. *Lancet* (7834):910, 1973.

4520 PRIMARY ACQUIRED SIDEROBLASTIC ANEMIA PRECEDING MONOCLONAL GAMMOPATHY AND MALIGNANT LYMPHOMA. (E.) Tranchida, L. (Wayne St.

U. Sch. Med., Detroit, Mich.), M. Palutke, M. D. Poulik and A. S. Prasad. *Am J Med* 55:559-564, 1973.

4521 ON THE HISTOMORPHOLOGY AND ORIGIN OF MALIGNANT CUTANEOUS CHANGES IN EPIDERMODYSPLASIA VERRUCIFORMIS. (E.) Ruiter, M. (Dept.

Dermatology, U. Groningen, Netherlands). *Acta Derm Venereol (Stockh)* 53(4):290-298, 1973.

4522 PROGESTERONE HIGH LEVELS ACTION ON DYSPLASIAS AND CARCINOMAS IN SITU OF THE ENDOMETRIUM. (E.) De Brux, J. (Paris, France) and

A. Schachter. *Acta Morphol Acad Sci Hung, Suppl* 14:113, 1973.

- 4523 CLONAL ORIGIN OF CHRONIC MYELOCYTIC LEUKEMIA. (E.) Barr, R. D. (Dept. Path., U. Aberdeen, Scotland) and P. J. Fialkow. *N Engl J Med* 289(6):307-309, 1973.
- 4524 THE PATHOGENESIS OF XANTHOMATA. (E.) Walton, K. W. (Dept. Exp. Path., U. Birmingham, England), C. Thomas and D. J. Dunkerley. *J Pathol* 109(4):271-290, 1973.
- 4525 ROENTGENOLOGICAL DIAGNOSIS OF EARLY STAGES OF GASTRIC CANCER. (Rus.) Barmin, V. S. (Dolgie Prudy Hosp., Moscow, USSR) and I. M. Pyltsov. *Vopr Onkol* 19(8):17-26, 1973.
- 4526 ON SPONTANEOUS BLASTOMOGENESIS IN MICE OVARIES. (Rus.) Gubareva, A. V. (USSR Ministry Public Hlth., Leningrad). *Vopr Onkol* 19(6):88-92, 1973.
- 4527 MALIGNANT TRANSFORMATION OF BLUE NEVI. (Rus.) Gordeladze, A. S. (Leningrad Med. Inst. Sanitation Hyg., USSR). *Vopr Onkol* 19(5):91-92, 1973.
- 4528 MORPHOGENESIS OF UTERINE MYOMAS. (Rus.) Serov, V. V. (I. M. Sechenov First Moscow Med. Inst., USSR), T. B. Zhuravleva, L. N. Vasilevskaya and Yu. G. Melnikov. *Akush Ginekol (Mosk)* (1):3-8, 1973.
- 4529 HISTOGENESIS OF ABRIKOSOV'S TUMOR [MYOBLASTOMA]. (Rus.) Apatenko, A. K. (N. N. Burdenko Central Military Clin. Hosp., USSR). *Ark Patol* 35(1):18-25, 1973.
- 4530 THE HISTOGENETIC STUDY OF MAMMARY FIBRO-ADENOMAS. (Fr.) Orcel, L. (Fac. Med., Paris, France) and D. Douvin. *Ann Anat Pathol (Paris)* 18(3):255-276, 1973.
- 4531 CANCER OF THE MAMMARY GLANDS WITH APOCRINE METAPLASIA. (Rus.) Paikova, L. V. (Leningrad Inst. Sanitation Hyg., USSR). *Ark Patol* 35(1):49-52, 1973.
- 4532 MORPHOGENESIS OF EXPERIMENTAL MYOMAS OF THE UTERUS. (Rus.) Zhuravleva, T. B. (I. P. Pavlov 1st Med. Inst., Leningrad, USSR) and Yu. G. Melnikov. *Ark Patol* 35(1):38-44, 1973.
- 4533 CYTOLOGICAL DIAGNOSIS OF REACTIVE CHANGES IN THE BRONCHIAL EPITHELIUM. (Rus.) Scherbakova, M. G. (USSR Ministry Publ. Hlth., Leningrad) and V. G. Rukavishnikova. *Vopr Onkol* 19(8):34-42, 1973.
- 4534 FOREIGN-BODY TUMORIGENESIS IN MICE: MOST PROBABLE NUMBER OF ORIGINATOR CELLS. (E.) Brand, K. G. (U. Minnesota Med. Sch., Minneapolis) L. C. Buoen and I. Brand. *J Natl Cancer Inst* 51(3):1071-1074, 1973.
- 4535 EXPERIMENTAL INVESTIGATIONS ON THE DIFFERENTIATION OF THE CARCINOMAS OF SKIN. (Ger.) Schnitzer, A. (No affiliation). *Dermatologica* 147(1):7-17, 1973.
- 4536 CLONAL EVOLUTION IN THE TERMINAL STAGE OF CHRONIC MYELOID LEUKEMIA. I. OBSERVATION OF STEM LINES WITH A METACENTRIC MARKER CHROMOSOME IN EIGHT PATIENTS. (Ger.) Bauke, J. (Hematol. Dept., U. Ulm, Germany). *Deutsch Med Wochenschr* 98(42):1956-1959, 1973.
- 4537 ISOZYME PATTERNS OF HEPATOMAS AND TUMOUR PROGRESSION. (E.) Weinhouse, S. (Temple U. Sch. Med., Philadelphia, Pa.). *Neoplasma* 20(5):559-562, 1973.
- 4538 THE HISTOGENESIS OF UTERINE LIPOMAS. (E.) Salm, R. (Roy. Cornwall Hosp., England). *Beitr Pathol* 149:284-292, 1973.
- 4539 RADIOLOGIC SEQUENCE FROM ESOPHAGITIS TO CARCINOMA. CASE REPORT. (E.) Canlas, E. M. (No affiliation) and M. Morgan. *Mo Med* 70(11):762-763, 767, 1973.
- 4540 ENZYME HISTOCHEMICAL STUDIES ON THE UTERINE CERVIX IN CEYLONESE WITH SPECIAL REFERENCE TO CERVICITIS AND CARCINOMA OF THE CERVIX. (E.) Panabokke, R. G. (Dept. Path., U. Ceylon, Peradeniya), B. Jayaweera, W. H. Fernando, D. E. Gunatilleka, A. M. Nandasena and W. B. Karunaratne. *Ceylon Med J* 17(3):137-146, 1972.
- 4541 THE MORPHOLOGICAL BEHAVIOUR OF THE PRESERVED BREAST TISSUE IN MAMMARY GLANDS WITH FIBROADENOMAS, FIBROSING ADENOSIS, EPITHELIOSIS, AND BREAST CARCINOMAS. (Ger.) Nizze, V. H. (Regional Hosp., Schwerin, Germany). *Arch Geschwulstforsch* 41(1):34-42, 1973.
- 4542 CONCERNING MORPHOLOGICAL CHARACTERISTICS OF THE ESOPHAGEAL MUCOUS MEMBRANE IN CHRONIC GASTRITIS AND CANCER OF THE STOMACH. (Rus.) Alexandrova, N. M. (Kazakh Res. Inst. Oncology, Radiology, Alma-Ata, USSR) and L. N. Inshakov. *Vopr Onkol* 19(6):31-34, 1973.
- 4543 THE MECHANISM OF THE ARISING OF NEOPLASMS IN THE RAT THYROID. (Fr.) Stoll, R. (Bergonie Fdn. Lab., Bordeaux, France), N. Faucounau, R. Maraud and D. Stoll. *C R Soc Biol (Paris)* 166(12):1654-1656, 1972.

4544 β -GLUCURONIDASE ACTIVITY OF THE EPITHELIAL CELLS AND STROMA CELLS IN PROSTATIC HYPERPLASIA. A BRIEF COMMUNICATION. (E.) Nilsson, T. (Dept. Urology, Path., St. U. New York, Buffalo), E. Schueller and W. Staubitz. *Invest Urol* 11(2): 145-148, 1973.

4545 THE DNA CONTENT IN THE GASTRIC MUCOSA EPITHELIUM IN CHRONIC GASTRITIS, POLYPOSIS AND CANCER. (A COMPARATIVE MICROSPECTROPHOTOMETRIC STUDY). (Rus.) Kazantseva, I. A. (USSR Acad. Med. Sci. Moscow). *Vopr Onkol* 19(9):51-54, 1973.

4546 THE LEVEL OF THE FOLLICLE-STIMULATING ACTIVITY IN PRECANCER AND CANCER OF THE MAMMARY GLAND. (Rus.) Iskakova, K. I. (KSSR Ministry Publ. Hlth., Alma-Ata, USSR) and N. N. Mezinova. *Vopr Onkol* 19(9):15-19, 1973.

See also:

* (Rev): 4213, 4223, 4231, 4237

- 4547 CANCER OF THE LARGE INTESTINE: EPIDEMIOLOGIC FINDINGS. (E.) Haenszel, W. (Nat'l. Inst. Hlth., Bethesda, Md.) and P. Correa. *Dis Colon Rectum* 16(5):371-377, 1973.

Epidemiologic findings on cancer of the large intestine are discussed, particularly regarding international comparisons, time trends, migrant populations and precursor lesions. The key epidemiologic clues appear to be an interrelationship between the magnitude of incidence rates, the sex-age patterns of incidence, and the anatomic localization of tumors, highlighted by the following features: 1) low male-female ratios for tumors above the sigmoid flexure in certain low-risk populations and male-female ratios close to unity in high-risk populations. 2) A rise in sigmoid/cecum ratios, steeper for males than for females, as one goes from low- to high-risk populations. 3) The male excess of colonic cancer among Japanese migrants to Hawaii. 4) The recent appearance in Connecticut of an upward displacement in male incidence for the cecum and ascending colon to approach the female level of risk. To account for these and other findings, the following model has been proposed. In low-risk populations where the disease is "endemic", colonic cancers are concentrated in the cecum and ascending colon, female cases are preponderant, and most of the rise to the maximum incidence level has occurred by age 50-55 yr. When a new etiologic factor is introduced into such a population, the transition from an "endemic" to an "epidemic" phase is first expressed as a rise in sigmoidal cancers among men more than 55 yr old. A rise in female sigmoidal cancer follows later, and the time lag is reinforced by a tendency for these female cases to appear at somewhat older ages than the male cases. As exposures to the etiologic factor become more intense and prolonged, a later phase is characteristically a rise in cecal and ascending colonic cancers more marked for males than for females, so that the female excess in cecal cancer prevailing under "endemic" conditions is diminished. The upward displacement in male risk may appear sequentially in time as involving successive segments of the colon, moving from the rectosigmoid junction to the cecum. The present observations on prevalence of adenomatous polyps suggest some link with the level of colonic cancer incidence in a given community, not only with respect to the magnitude of prevalence but also with respect to distribution by age, sex, anatomic localization, and multiplicity.

- 4548 TROPHOBLASTIC DISEASE IN UGANDA. (E.) Leighton P. C. (Mulago Hosp., Kampala, Uganda). *Am J Obstet Gynecol* 117(3):341-344, 1973.

In the four yr period from 1967 to 1970, 243 cases of trophoblastic disease were recorded by the Department of Pathology, Makerere University Medical School, Kampala, Uganda. The incidence of hydatidiform mole at the country's major hospital was 1.03 cases/1,000 deliveries over 28 wk of gestation. This incidence is similar to that observed in Europe. Fifty-two cases of choriocarcinoma were recorded in the same four yr period, most cases being concentrated in Kampala. Of the nonmolar deliveries, 5.4% of the

total women were 35 yr of age or older; yet, 32.5% of the total choriocarcinoma cases and 22% of the total hydatidiform mole cases were from this age group.

- 4549 INCIDENCE OF MALIGNANT MELANOMA OF THE SKIN IN NORWAY, 1955-1970. VARIATIONS IN TIME AND SPACE AND SOLAR RADIATION. (E.) Magnus, K. (Cancer Registry Norway, Oslo). *Cancer* 32(5):1275-1286, 1973.

Variations in the incidence of malignant melanoma of the skin in Norway were analyzed from 2541 cases occurring between 1955 and 1970. During the 15 year period, the age-adjusted incidence rate has more than doubled for both sexes. A marked north-south gradient was observed, the incidence in southern Norway being almost 3 times greater than that in the northern part of the country. Melanomas of the neck-trunk and lower limb are more common in the capital and provincial towns than in rural areas; melanomas of the face and foot deviate from this pattern. The incidence of melanoma of the neck-trunk region is higher among males, while the reverse is true for melanomas of the lower and upper limb; melanomas of the face and foot showed no variation with sex. Melanomas of the face has increased slightly with time, while there has been a manifold increase in the incidence of melanomas of the neck-trunk and lower limb; an intermediate trend was observed for the upper limb and other sites except the foot, which has showed no systematic variations. Large cohort variations between those born around the turn of the century and those born around 1935, with the incidence being higher among the younger generations. It is concluded that exposure to sunlight is an important factor in the etiology of malignant melanoma of the skin. The effect of exposure appears to be local. No support is given to the hypothesis of a systemic effect.

- 4550 EPIDEMIOLOGICAL AND PATHOLOGICAL OBSERVATIONS ON RETICULUM CELL SARCOMA IN (C57BL/Cne x C3H/Cne)_{F1} MICE. (It.) Covelli, V. (Lab. Animal Radiobiol., Natl. Committee Nuclear Energy, Rome, Italy), P. Metalli, B. Bassani, B. di Caterino and G. Silini. *Tumori* 59(2):97-118, 1973.

Long-term observations on untreated animals have shown that spontaneous reticulum cell sarcomas (RCS) developed in 56.5% male mice of the hybrid (C57BL/Cne x C3H/Cne)_{F1} strain. The average age at death of mice with tumors was 949 days, compared to 929 days for all causes and no age-specific peak of mortality occurred over the entire life span of the animals. The spleen and all the lymph nodes, including the mesenteric nodes, were always invaded. Neoplastic growth occurred less frequently in kidneys, liver and lungs (77, 70 and 40%, resp.); only occasionally in other organs such as adrenals and testes; and never in the thymus. The tumor consisted primarily of highly undifferentiated reticular cells, typically proliferating from the periarteriolar region of the lymph follicles in

the spleen. This suggests that RCS in this strain of mice may be classified as type A according to Dunn. Electron microscope observations showed the presence of a few virus-like particles, both in tumor cells and in sediment from cell-free extracts. Transplantation of cells from spontaneous RCS into both normal and lethally-irradiated syngeneic recipients was successful only in 4 out of 7 trials, regardless of the tissue of origin of the neoplastic cells (spleen, lymph nodes or bone marrow). Virus-like particles occurred more frequently in transplanted tumors. Inoculation of cell-free extracts into neonatal mice of low-leukemia strains has not so far been successful. Splenectomy of young animals and i.v. injection of syngeneic bone marrow cells immediately following a lethal dose of whole-body x-irradiation significantly reduced the frequency of spontaneous tumors.

4551 CELL PROLIFERATION IN RECTAL CARCINOMA AND RECTAL MUCOSA. A STATHMOKINETIC STUDY.

(E.) Camplejohn, R. S. (Dept. Pathol. Surg., U. Newcastle upon Tyne, England), G. Bone and W. Aherne. *Eur J Cancer* 9(8):577-581, 1973.

A stathmokinetic technique, involving injection of vincristine sulfate (0.045 mg/kg, i.v.) to block cells in metaphase, was used *in vivo* to estimate the rate of cell proliferation in a group of 19 carcinomas of the large bowel. This rate of proliferation was compared with that found using the same technique on normal rectal mucosa from seven tumor-bearing patients. The mean apparent cell cycle time ($t_c(a)$) of the tumors was estimated at 192 hr, and that of the normal rectal mucosa at 82 hr. The duration of mitosis (t_m) was 2.3 hr in tumor cells and 1.2 hr in normal rectal mucosal cells. Speculative estimates of the rate of cell loss and of growth fraction in the tumors are made by combining these results with those obtained by other workers using different kinetic techniques. It is suggested that decrease in cell loss rather than an increase in proliferation is responsible for the progressive growth of tumors. Since most anticancer drugs act only on proliferating cells, chemotherapy will have limited success unless normal tissues are protected against the action of these compounds.

4552 NON-HODGKIN'S LYMPHOMA IN CHILDHOOD: EPIDEMIOLOGIC FEATURES. (E.) Grundy, G. W. (Natl. Cancer Inst., Bethesda, Md.), E. T. Creagan and J. F. Fraumeni, Jr. *J Natl Cancer Inst* 51(3):767-776, 1973.

Death certificates of 2642 U.S. children who died of non-Hodgkin's lymphoma, 1960-67, and hospital charts of 900 children with these neoplasms were studied. Lymphosarcoma showed the highest morbidity throughout childhood, with small age peaks at 3-5 years for both sexes. In contrast, reticulum cell sarcoma increased gradually with age.

For all cell types combined, non-Hodgkin's lymphoma was 2 1/2 times more common among males than females under age 15. The highest sex ratio characterized lymphosarcoma of the gastrointestinal tract (6:1 male excess). As a feature shared by Hodgkin's disease in childhood, male preponderance seems to reflect a sex-related susceptibility common to all forms of solid lymphoreticular neoplasms. Transitions to adult age and sex patterns occurred in late childhood. There was no geographic or temporal variation in mortality to suggest major environmental influences. Mortality time trends between 1950-69 revealed a significant decline in lymphosarcoma and increase for reticulum cell sarcoma and other cell types. In the hospital series, a few children had altered immune states, celiac malabsorption, or radiation exposure. Sibs of children with non-Hodgkin's lymphoma showed an increased risk for lymphoma, leukemia, and brain neoplasms. The frequency of leukemic transformation in non-Hodgkin's lymphoma was greatest in early childhood, affecting 34% of children under 5 years. Epidemiologic comparison between childhood lymphoma and leukemia showed similarities as well as important differences.

4553 TESTICULAR TUMORS: EPIDEMIOLOGIC, ETIOLOGIC, AND PATHOLOGIC FEATURES. (E.)

Mostofi, F. K. (Armed Forces Inst. Path., Washington, D.C.). *Cancer* 32(5):1186-1201, 1973.

Epidemiologic, etiologic, and pathologic findings based on the observation of 7,000 testicular tumors in humans are reported. Testicular tumors constitute the fourth most common cause of death from neoplasia in the 15-34-year age group. They are rare among Negroes and in Asia and New Zealand; although they occur in animals, there is considerable species differentiation. Testicular tumors appear to occur more frequently: in conjunction with cryptorchidism; in infancy, between ages 20 and 35 years, and at age 50 years and older; in persons presenting a history of trauma, orchitis, or endocrine abnormalities; among those who have had testicular tumors or whose close relatives have; and in those with dysgenetic testes. Testicular tumors have also been reliably produced in experimental animals via chemical injection or transplantation. Germ cell tumors comprise the majority of testicular tumors and present one or more of four basic histologic patterns: seminoma, embryonal carcinoma, choriocarcinoma, and/or teratoma. About 40% of all tumors are admixtures of two or more of these basic types. Tumors of specialized gonadal stroma constitute about 6% of all testicular tumors. They include tumors of the Leydig cells, Sertoli cells, and/or granulosa-theca cells. The most common of the metastatic tumors, initially manifested as testicular neoplasia, is the malignant lymphoma; it constitutes about 1% of all testicular tumors. Most malignant adnexal tumors are sarcomas, the most common being the rhabdomyosarcomas which occur in persons from 5 months to 28 years of age. The gross and histologic features of all of these tumors are discussed.

- 4554 COMPUTER ANALYSIS OF TRACER KINETIC DATA FROM A HUMAN HEMATOPOIETIC CELL LINE. (E.) Fried, J. (Sloan-Kettering Inst., New York, N.Y.), S. Zietz, A. Todo, A. Strife and B. Clarkson. *Proc Am Assoc Cancer Res* 13(March):16, 1972.
- 4555 MATHEMATICAL DESCRIPTION OF THE GROWTH CHARACTERISTICS AND CELLULAR KINETICS OF 3 RAPID-, 3 INTERMEDIATE-, AND 3 SLOW-GROWING HEPATOMAS. (E.) Looney, W. B. (U. Virginia Sch. Med., Charlottesville), F. B. Lillich, A. A. Mayo, P. Allen, J. Morrow and H. P. Morris. *Proc Am Assoc Cancer Res* 13(March):66, 1972.
- 4556 CHARACTERIZATION OF THE GROWTH KINETICS OF SUBCUTANEOUS AND METASTATIC LEWIS LUNG CARCINOMA. (E.) Simpson-Herren, L. (Southern Res. Inst., Birmingham, Ala.). *Proc Am Assoc Cancer Res* 13(March):24, 1972.
- 4557 HISTOCHEMICAL, MORPHOLOGIC, AND GROWTH CHARACTERISTICS OF A HUMAN NEUROBLASTOMA. (E.) Barth, R. F. (U. Kansas Med. Ctr., Kansas City), D. R. Dunn and D. J. Svoboda. *Proc Am Assoc Cancer Res* 13(March):22, 1972.
- 4558 CHANGES IN CELL PROLIFERATION RATE IN THE MOUSE UTERINE EPITHELIUM DURING CONTINUOUS OESTROGEN TREATMENT. (E.) Lee, A. (Imperial Cancer Res. Fund, London, England). *Acta Endocrinol* 74(2):220, 1973.
- 4559 INCIDENCE OF TUMORS IN THE NERVOUS SYSTEM IN HONG KONG. (E.) Wen, H. L. (Kwong Wah Hosp., Kowloon, Hong Kong) and S. Y. C. Cheung. *Int Surg* 58(8):555-556, 1973.
- 4560 INCIDENCE AND BEHAVIOR PATTERN OF INTRACRANIAL TUMORS IN CEYLON. (E.) Weinman, D. F. (Gen. Hosp., Colombo, Ceylon). *Int Surg* 58(8):548-554, 1973.
- 4561 INTERRELATIONSHIPS BETWEEN SILICOSIS AND MALIGNANT NEOPLASMS. (Rus.) Mokronosova, K. A. (Inst. Industrial Hyg. Occupational Dis., Sverdlovsk, USSR), N. K. Shabynina, B. A. Katsnel'son and D. M. Zislin. *Vopr Onkol* 19(5):3-7, 1973.
- 4562 ESOPHAGEAL CANCER IN CALIFORNIA 1942-1969: THE CALIFORNIA TUMOR REGISTRY EXPERIENCE. (E.) Krain, L. S. (Med. Ctr., U. California, Los Angeles). *J Surg Oncol* 5(3):267-275, 1973.
- 4563 SOME ANAMNESIS DATA OF PATIENTS WITH UTERINE CERVIX AND CORPUS CARCINOMA (SERIES OF TWENTY YEAR PERIOD 1952-1971). (E.) Szamborski, J. (Med. Sch. Warsaw, Poland), E. Klosinka-Kita, I. Roszkowski and M. Liebhart. *Neoplasma* 20(4):427-435, 1973.
- 4564 PRIMARY LIVER CANCER IN MAN AS A POSSIBLE SHORT DURATION SEASONAL CANCER. (E.) Purves, L. R. (Life Sci. Div., Atomic Energy Board, Pretoria, S. Africa). *S Afr J Sci* 69(6):173-178, 1973.
- 4565 CANCER PATTERNS IN AFRICANS OF SOUTHERN AFRICA. (E.) Anonymous. *S Afr J Sci* 69(6):164-166, 1973.
- 4566 CARCINOMA OF THE BREAST IN WOMEN 30 YEARS OF AGE OR LESS. (E.) Birks, D. M. (British Columbia Cancer Inst., Vancouver), G. M. Crawford, L. G. Ellison and F. R. Johnstone. *Surg Gynecol Obstet* 137(1):21-25, 1973.
- 4567 DISABILITY DUE TO NEOPLASMS IN DRIVERS. (Rus.) Smulevich, V. B. (Res. Inst. Labor Hygiene, Occupational Dis., Gorjky, USSR), A. I. Vaisman and M. Sh. Ixanov. *Vopr Onkol* 19(8):57-61, 1973.
- 4568 THE EFFECT OF ABDOMINAL PARACENTESIS ON DYNAMICS OF THE ASCITIC TUMOR GROWTH. (Rus.) Vasiljeva, G. S. (Kazakh Int. Oncology, Radiology, Alma-Ata, USSR) and M. L. Efimov. *Vopr Onkol* 19(8):90-93, 1973.
- 4569 CIGARETTE SMOKING AND EXPOSURE TO OCCUPATIONAL HAZARDS. (E.) Friedman, G. D. (Permanente Med. Group, Oakland, Calif.), A. B. Siegel and C. C. Seltzer. *Am J Epidemiol* 98(3):175-183, 1973.
- 4570 ON EPIDEMIOLOGY OF ESOPHAGEAL CANCER IN BURYAT ASSR. (Rus.) Dulganov, K. P. (Buryat Repub. Oncol. Dispensary, U.S.S.R.). *Vopr Onkol* 19(6):103-107, 1973.
- 4571 INCIDENCE OF CHILDHOOD LEUKAEMIA. (E.) Falk, H. (Ctr. Disease Control, Atlanta, Ga.), C. W. Heath, Jr., D. J. Fernbach and J. M. Falletta. *Lancet* (7833):862, 1973.
- 4572 THE INCIDENCE OF CANCER IN MARRIED COUPLES. (E.) Walach, N. (Hadassah-Hebrew U. Med. Ctr., Jerusalem, Israel) and Y. Horn. *JAMA* 226(2):201, 1973.
- 4573 INTRACRANIAL TUMORS IN INDIA: INCIDENCE AND VARIATIONS. (E.) Ramamurthi, B. (Madras Med. Coll., India). *Int Surg* 58(8):542-546, 1973.
- 4574 INTRACRANIAL NEOPLASMS IN MALAYSIA. (E.) Selby, R. (Cook Cty. Hosp., Chicago, Ill.) and N. Pereira. *Int Surg* 58(8):536-541, 1973.

- 4575 THE MEDICAL UNIVERSITY OF SOUTH CAROLINA TUMOR REGISTRY. (E.) Miller, M. C., III. (Med. U. South Carolina, Columbia), P. H. O'Brien, M. T. Mangum and H. L. Hyer. *J SC Med Assoc* 69(8): 285-291, 1973.
- 4576 KINETIC PRINCIPLES OF GROWTH OF TRANSPLANTABLE HEPATOMAS WITH DIFFERENT DEGREES OF DIFFERENTIATION. (E.) Bogdanov, G. N. (Acad. Sci., USSR), V. M. Shmonina and N. M. Emanuel. *Bull Exp Biol Med* 75(3):310-312, 1973.
- 4577 ASBESTOS HAZARDS. (E.) Fritsch, A. J. (Ctr. Sci. Public Interest, Washington, D.C.) and B. Castleman. *J Air Pollut Control Assoc* 23(9):807, 1973.
- 4578 WORKER EXPOSURE TO CARCINOGENS DEBATED. (E.) Anonymous. *Chem Eng News* 51(38): 5, 1973.
- 4579 CHILDHOOD LEUKAEMIA IN MANCHESTER. (E.) Leck, I. (U. Dept. Community Med., Oncology, Child Hlth., Path., Manchester, England), P. M. Jones, J. K. Steward, D. I. K. Evans and H. B. Marsden. *Lancet* 2(7827):509-510, 1973.
- 4580 PROSTATIC CARCINOMA AT AUTOPSY IN HIROSHIMA AND NAGASAKI JAPANESE. (E.) Bean, M. A. (Atomic Bomb Casualty Commission, Hiroshima, Japan), R. Yatani, P. I. Liu, K. Fukazawa, F. W. Ashley and S. Fujita. *Cancer* 32(2):498-506, 1973.
- 4581 CANCER AND OTHER NON-INFECTIVE DISEASES OF THE BOWEL. EPIDEMIOLOGY AND POSSIBLE CAUSATIVE FACTORS. (E.) Burkitt, D. P. (Med. Res. Council, London, England). *Rend Gastroenterol* 5(1): 33-39, 1973.
- 4582 LARYNGEAL PAPILLOMA: ETIOLOGIC AND THERAPEUTIC CONSIDERATIONS. (E.) Cook, T. A. (Baylor Coll. Med., Houston, Tex.), A. M. Cohn, J. P. Brunschwig, H. Goepfert, J. S. Butel and W. E. Rawls. *Ann Otol Rhinol Laryngol* 82(5):649-655, 1973.
- 4583 PROLIFERATION OF TRACHEAL EPITHELIAL CELLS IN NORMAL AND VITAMIN A-DEFICIENT SYRIAN GOLDEN HAMSTERS. (E.) Harris, C. C. (Natl. Inst. Hlth., Bethesda, Md.), T. Silverman, J. M. Smith, F. Jackson and H. G. Boren. *J Natl Cancer Inst* 51(3):1059-1062, 1973.
- 4584 KINETICS OF LYMPHOCYTES IN CHRONIC LYMPHOCTIC LEUKEMIA: STUDIES USING CONTINUOUS ³H-THYMIDINE INFUSION IN TWO PATIENTS. (E.) Theml, H. (Municipal Hosp., Munich-Schwabing, Germany), F. Trepel, P. Schick, W. Kaboth and H. Begemann. *Blood* 42(4):623-633, 1973.
- 4585 CERVICAL CANCER MORTALITY STUDY. A PRELIMINARY REPORT. (E.) Cervical Cancer Mortality Sub-Committee. *Minn Med* 56(10):9-11, 1973.
- 4586 KINETICS OF LYMPHOCYTES IN HODGKIN'S DISEASE. (Ger.) Schick, P. (Municipal Hosp., Munich-Schwabing, Germany), F. Trepel, H. Theml, S. Benedek, P. Trumpp, W. Kaboth, H. Begemann and T. M. Fliedner. *Blut* 27(4):223-235, 1973.
- 4587 AN EPIDEMIOLOGICAL STUDY OF CENTRAL NERVOUS SYSTEM NEOPLASMS IN OKLAHOMA. (E.) Mackenthun, A. V. (U. Oklahoma Coll. Hlth., Norman), N. R. Asal and P. S. Anderson, Jr. *J Okla St Med Assoc* 66(10):417-423, 1973.
- 4588 MORTALITY FROM CANCER OF THE OESOPHAGUS IN BRITAIN. (E.) Tuyns, A. J. (Internat. Agency Res. Cancer, Lyon, France) and L. M. F. Masse. *Int J Epidemiol* (2):241-245, 1973.
- 4589 ASSESSMENT OF SIGNIFICANCE OF PROPORTIONS OF INTRADUCTAL AND INFILTRATING TUMOR GROWTH IN DUCTAL CARCINOMA OF THE BREAST. (E.) Silverberg, S. G. (U. Colorado Sch. Med., Denver) and A. R. Chitale. *Cancer* 32(4):830-837, 1973.
- 4590 WEIBULL DISTRIBUTIONS FOR CONTINUOUS-CARCINOGENESIS EXPERIMENTS. (E.) Peto, R. (Radcliffe Infirm., Oxford U., England) and P. Lee. *Biometrics* 29(3):457-470, 1973.
- 4591 EPIDEMIOLOGY OF PLEURAL MESOTHELIOMA IN URBAN INDUSTRIAL AREAS. (It.) Rubino, G. F. (Inst. Industrial Med., U. Turin, Italy), G. Scansetti and A. Donna. *Med Lav* 63(7-8): 299-315, 1972.

See also:

- * (Rev): 4209, 4214, 4217, 4233, 4234
 * (Immun): 4481

- 4592 METABOLIC REGULATORS IN THE PLACENTA AND THEIR EFFECT ON NORMAL AND TUMOR CELLS. (Ger.) Letnansky, K. (Inst. Cancer Res., U. Vienna, Austria). *Exp Pathol* 8(3):205-212, 1973.

The effect of extracts of the deciduous membranes and chorion from bovine placentas was investigated on respiration and incorporation of amino acids into polypeptides in Ehrlich ascites tumor cells, rat liver slices, and cell-free rat liver homogenates. Extracts of deciduous membranes increased respiration by 30% in Ehrlich ascites tumor cells but inhibited respiration in rat liver slices by at least 20%. Chorionic extracts inhibited amino acid incorporation in Ehrlich ascites tumor cells but increased it in rat liver microsomes; extracts of deciduous membranes inhibited amino acid incorporation in both systems. These findings and the results of previous investigations on the effect of placental extracts on phosphate incorporation indicate that factors are present in the maternal part of the placenta which intensify catabolic processes in tumor cells and anabolic processes in normal liver cells. Separation of these extracts by gel electrophoresis on Sephadex G-100 revealed that chorionic extract contains at least seven different factors which stimulate respiration and amino acid incorporation in rat liver microsomes, while extracts of deciduous membranes also contain two components which inhibit amino acid incorporation. The effects of these stimulators and inhibitors were proportional to the quantity of them which was added to the system. By means of electrophoresis on SDS polyacrylamide gel it was demonstrated that the molecular weights of stimulators ranged from 10,000 to 60,000 daltons, while one of the two inhibitors contained proteins with molecular weights of 10,000 and 17,000 daltons. It is possible that all fractions obtained on gel electrophoresis are completely different components, but it is far more likely that they are subunits having different molecular weights, metabolically active fragments of a few basic components, or one or more active components associated with peptide carriers of different molecular weights.

- 4593 SQUAMOUS-CELL CARCINOMA OF THE ANUS. (E.) Failes, D. (Sydney Hosp., Australia) and B. P. Morgan. *Dis Colon Rectum* 16(5):397-401, 1973.

A retrospective study was conducted on 76 patients treated by different methods from 1947 to 1967 for squamous-cell carcinoma of the anus at either Sydney Hospital or Royal Prince Alfred Hospital. The tumors were divided into two groups based on anatomical location: 11 had "anal-margin" carcinoma arising below the pectinate line and the remainder had "anal-canal" tumors arising on or above the pectinate line. Six of nine anal-margin patients available for follow-up were alive after 5 yr as opposed to 16 of 50 anal-canal tumor patients, suggesting an overall more favorable prognosis for the former group. Some form of curative treatment was attempted in a total of 53 cases. Primary radiotherapy and conservative local excision were highly unsuccessful with only 3 of 17 of the former and only 1 of 7 of the latter being regarded as

tumor-free after 5 yr. Although only 8 of 19 patients treated by radical surgery were alive after 5 yr, this appeared to be the only procedure which offered a reasonable prospect of cure.

- 4594 RNA-DIRECTED DNA-POLYMERASE ACTIVITY IN RETINOBLASTOMA: REPORT OF ITS PRESENCE AND POSSIBLE SIGNIFICANCE. (E.) Albert, D. M. (Yale U. Sch. Med., New Haven, Conn.) and T. W. Reid. *Trans Am Acad Ophthalmol Otolaryngol* 77(5):631-640, 1973.

The presence of RNA-directed DNA-polymerase in human ocular tumors, certain related tumors, and control tissues was investigated. Using known techniques, RNA-directed DNA-polymerase type activity was found to be present in ten specimens of retinoblastoma. This enzyme activity was also found in medulloblastoma and neuroblastoma. This appears to be the first demonstration of an RNA-directed DNA-polymerase activity in a human ocular tumor. This enzyme was not demonstrated in ocular tissues, brain or adrenal gland, in a variety of other malignant tumors, including lymphomas and sarcomas, or in certain rapidly dividing tissue culture cell lines. Additional control studies were carried out using avian myeloblastosis virus and feline sarcoma and lymphoma virus and experimental retinal tumors induced with feline sarcoma virus in cats. The biochemical activity of the viral enzyme in these materials corresponded closely to that in the human tumors.

- 4595 COMPARISON OF CELL COAT ACID MUCOPOLYSACCHARIDES OF NORMAL LIVER AND VARIOUS ASCITES HEPATOMA CELLS. (E.) Yamamoto, K. (Zool. Inst., U. Tokyo, Japan) and H. Terayama. *Cancer Res* 33(10):2257-2264, 1973.

Cell coat acid mucopolysaccharides were released from *in vivo* ^3H -glucosamine prelabeled normal rat liver and ascites hepatoma cells of various strains by mild *in vitro* papain treatment. The mucopolysaccharide fractions from the different cell types were separated by Sephadex G-50 gel filtration and were compared. The macromolecular peak fractions thus obtained from normal cells contained about 2% of the total cellular radioactivity and about 50% of the membrane-associated label. These fractions stained metachromatically with toluidine blue, showed a mucin clot reaction with polycations, contained sulfuric acid in the HCl hydrolysate, and were resistant to hyaluronidase and chondroitinase treatment. Although the peak fractions from prelabeled ascites hepatoma cells contained a higher percentage (10-18%) of the total cellular label than did normal liver cells, their gel filtration patterns were, in general, similar. These fractions, however, unlike those from the normal liver cells, were partially degraded by chondroitinase. The degree of degradation differed from one hepatoma strain to another and ranged from 2.7% for AH-371-A cells to 50.4% for AH-130 F(N) cells. The macromolecular material released from normal cells by papain migrated as a single band on cellulose acetate strips under various electrophoretic conditions. Corresponding materials from the various hepatoma cells showed multiple components with different electrophoretic mobilities. The liver cell coat material was elec-

trophoretically distinct from all the reference acid mucopolysaccharides used.

- 4596 MITOSIS IN TISSUE CULTURES OF HUMAN GIANT CELL TUMORS OF BONE. (E.) Troise, G. D. (Dept. Res., Angel H. Roffo Inst. Oncology, Buenos Aires, Argentina), E. S. de Lustig, F. Schajowicz and H. Gallardo. *Oncology* 28(3): 193-203, 1973.

In vitro tissue cultures of 12 human osteoclastomas and 2 benign chondroblastomas were studied histopathologically using a variety of stains. Among the mononucleated cells of the histiocytic and fibroblastic type, were many binucleated and multinucleated giant cells. The nuclei of the mono- and binucleated cells were scanty in chromatin with two or more nucleoli and a clear cytoplasm; many were at different stages of mitosis. Besides normal mitosis, arrangements of the tripolar, tetrapolar, and multipolar type were seen; multipolar arrangements were also observed with irregular chromosome arrays. Some of the multinucleated cells seem to be the result of the confluence of mononucleated cells. It is possible that the tumoral multinucleated cells arise in an earlier stage from mono- or binucleated cells by mitotic or amitotic division. Multipolar mitosis, and eventually the confluence of mononucleated cells, would originate syncytia with a small number of nuclei; then, or in a subsequent stage, the syncytia would grow by recruiting neighboring cells.

- 4597 DIFFERENCES IN THE EFFECT OF 4-HYDROXY-PENTENAL ON SUCCINATE OXIDATION AND SUCCINATE DEHYDROGENASE IN LIVER AND TUMOR CELLS. (Ger.) Zollner, H. (Inst. Biochem., Karl Franz U., Graz, Austria) and E. Schauenstein. *Z Krebsforsch* 79(2):108-113, 1973.

ADP-stimulated succinate oxidation and succinate dehydrogenase activities were studied *in vitro* in Ehrlich ascites tumor cells from NMRI mice, in liver slices from normal male Wistar rats, and in isolated mitochondria from these cells by incubating them for 30 min at 37 C with $10^{-3}M$ of 4-hydroxypentenol (HPE). Succinate dehydrogenase was determined by the reduction of 3-(4,5-dimethylthiazolyl)-2,5-diphenyl tetrazolium bromide. HPE inhibited ADP-stimulated succinate oxidation in Ehrlich ascites tumor cells by an average of 75% but had no effect on succinate oxidation in normal liver cells. However, HPE inhibited ADP-stimulated succinate oxidation to the same extent in mitochondria isolated from Ehrlich ascites tumor cells and those isolated from normal rat liver. Similarly, HPE inhibited succinate dehydrogenase activity to about the same extent in mitochondria isolated from Ehrlich ascites tumor cells than in those isolated from normal rat liver cells, while succinate dehydrogenase in intact tumor cells was inhibited to a far greater extent than in normal rat liver cells. These findings demonstrate that inhibition of enzymes by HPE in tumor cells but not in normal liver cells is not due to differences in enzyme sensitivities to HPE,

but is the result of extramitochondrial factors which protect mitochondrial enzymes. It is suggested that cytoplasmic glutathione and alcohol dehydrogenase in normal liver cells react with HPE and prevent it from entering the mitochondria.

- 4598 CARCINOMA ASSOCIATED WITH RENAL TUBERCULOSIS: A REPORT OF TWO CASES, WITH A BRIEF REVIEW OF THE LITERATURE. (E.) Mehrotra, M. L. (Inst. Med. Sci., Banaras Hindu U., Varanasi, India), I. M. Gupta, N. N. Khanna and S. Gupta. *Aust N Z J Surg* 43(1):58-60, 1973.

Two cases, a 40-year-old female and a 45-year-old male, of primary renal carcinoma associated with tuberculosis of the same kidney are reported. Both patients presented with flank mass and pain with hematuria, and the provisional clinical diagnosis in each case was renal carcinoma. Histologically, both tumors consisted of granular cells arranged in solid alveoli, sheets, and papillary processes with abundant cytoplasm, regular and hyperchromatic nuclei, and scanty stroma. Tuberculous granulomata were abundant. Only 26 similar cases are reported in the world literature, and the association is rare even in countries where both diseases are relatively common. These findings lend support to the hypothesis that the presence of tuberculosis renders an organ less susceptible to the development of cancer because of nonspecific stimulation of the reticuloendothelial system.

- 4599 EXTRA-ABDOMINAL AND ABDOMINAL DESMOIDS IN THE SAME PATIENT. (E.) Musiatowicz, B. (Med. Sch., Bialystok, Poland), L. Ostrowski and M. Grygoruk. *Neoplasma* 20(4):471-475, 1973.

A case of a 25 yr old woman in whom extra-abdominal and abdominal desmoids occurred one after another is described. The extra-abdominal desmoids were associated with two subclavicular tumors occurring within 11 months of each other. Microscopically the tumors were composed of fibroblasts, elongated spindle-shaped cells, forming fascicles and bands which interlaced and often merged. Collagen bands were visible between the cells. Multiple destroyed muscle bands and giant polynuclear muscle cells were observed at the periphery where the lesions infiltrated the muscle. An abdominal tumor, diagnosed 11 months after the second subclavicular tumor, was located within the sheath of the left abdominal rectus at its upper end. The tumor had all the features of fibromatosis abdominalis (desmoid).

- 4600 CHANGES IN THE COMPOSITION OF LIPID FRACTIONS FROM CELLS OF GROWING EHRlich ASCITES CARCINOMA. (Rus.) Lankin, V. Z. (Inst. Chem. Phys., Moscow, USSR). *Dokl Akad Nauk SSSR* 211(2):486-487, 1973.

Phosphatides, triglycerides, free and esterified cholesterol, and free fatty acids were determined in chloroform-methanol extracts from Ehrlich ascites carcinoma during its growth in noninbred male white mice. The ascites carcinoma had been transplanted

after seven days of development. During the first few days the phosphatide and free cholesterol contents of the tumor cells increased, while the triglyceride content decreased; the contents of these fractions remained almost unchanged from day five on. The free fatty acid content decreased in tumor cells, reaching a minimum 10 days after transplantation when tumor growth was at a maximum, while the free fatty acid content of the ascites fluid increased 2.5 times from 7 to 14 days after tumor transplantation. During the early stages of tumor development larger quantities of these lipid fractions were present in tumor cells than in normal tissue. From 2 to 16 days after transplantation the relative content of phosphatides in tumor cells increased about 1.5 times, while the relative free fatty acid and triglyceride contents decreased to about 1/3 and 1/2 of their initial values, resp. The relative content of esterified cholesterol remained almost unchanged. These findings support the hypothesis that the host mobilizes lipids to supply developing tumors.

- 4601 KARYOTYPIC VARIATION IN BENIGN PLEOMORPHIC ADENOMA OF THE PAROTID AND IN NORMAL SALIVARY GLANDS. (E.) Scappaticci, S. (Euratom Unit Human Radiation Cytogenetics, U. Pavia, Italy), F. Lo Curto and E. Mira. *Acta Otolaryngol* 76(2-3):221-228, 1973.

Numerical and structural karyotypic variation was found in cells cultured *in vitro* from six benign mixed tumors (pleomorphic adenoma) of the human parotid gland. Three submaxillary glands with chronic sialadenitis and six histologically normal salivary glands from subjects with neoplasms of the upper aero-digestive tract were studied originally as controls, but found to display a similar degree of karyotypic variation, mainly due to gain and/or loss of -E- and -G-like chromosomes. Normal karyotypes were found in the normal salivary glands from two subjects with other conditions. The hypothesis is put forward that salivary gland tissue is liable to karyotypic variation due to the presence of some factor(s) *in vivo* and/or its activation *in vitro*. This unknown *in vivo* factor(s) may be connected with the presence of the abnormal condition itself.

- 4602 DISTRIBUTION AND CONCENTRATION OF ZINC IN THE SUBCELLULAR FRACTIONS OF BENIGN HYPERPLASTIC AND MALIGNANT NEOPLASTIC HUMAN PROSTATE. (E.) Dhar, N. K. (Central Drug Res. Inst., Lucknow, India), T. C. Goel, P. C. Dube, A. R. Chowdhury and A. B. Kar. *Exp Mol Pathol* 19(2):139-142, 1973.

The subcellular distribution of zinc in normal and diseased prostate tissue was determined by polarography. Total zinc content was 540.0 µg/g in normal tissue, 746.4 µg/g in benign hyperplastic prostate, and 168.2 µg/g in malignant neoplastic prostate. The pattern of distribution of the metal in the subcellular fractions (nuclear > mitochondria > microsomes > supernatant) was, however, unaltered, except that in the malignant neoplastic tissue the concentration in the nuclear and the mitochondrial fractions was virtually equal. This subtle change in distribution

pattern could be related to the transition from benign hyperplastic status to frank malignant neoplasia. It is conceivable that cancer disturbs the binding of prostate zinc to its carrier protein, resulting in markedly diminished concentration; conversely, some factor in benign overgrowth may promote increased binding of the metal to protein, thus leading to high overall concentration.

- 4603 INHIBITION OF SOME DNA POLYMERASE ACTIVITIES FROM CULTURED BURKITT CELLS BY THIOLATED RIBONUCLEIC ACIDS. (E.) Srivastava, B. I. S. (Dept. Exp. Ther., Roswell Park Mem. Inst., Buffalo, N.Y.) and T. J. Bardos. *Life Sci* [1] 13(1):47-53, 1973.

Unmodified poly C and unmodified ribosomal RNA showed little (< 20%) or no inhibition of 6-7S cytoplasmic, 3-4S cytoplasmic, and 3-4S chromatin-associated DNA-directed DNA polymerases and of RNase-sensitive endogenous DNA polymerase and DNA-directed DNA polymerase activity associated with a particulate material (p = 1.16-1.18 g/ml) from Burkitt cells. However, thiolated poly C and thiolated RNA preparations, in which 9.5% and 2%, resp., of all cytosine and uracil bases in the polynucleotide molecule had been converted to the corresponding 5-mercapto-pyrimidine derivatives, were strongly inhibitory (70-97%). Moreover, the thiolated nucleic acids were more inhibitory to 6-7S enzyme than to 3-4S enzyme. Thiolation of nucleic acids thus appears to be a potentially important procedure for the development of agents which may be selective against certain polymerases.

- 4604 BUFFY-COAT LEUCOCYTES IN HODGKIN'S DISEASE. (E.) Kesselman, M. (Hlth. Sci. Ctr., Winnipeg, Manitoba, Canada), A. Sasyniuk and W. Hryniuk. *Lancet* (7835):977, 1973.

The buffy coats of blood from 30 patients with Hodgkin's and non-Hodgkin's lymphomas and from 10 non-lymphoma patients were examined for the presence of abnormal looking basophilic cells similar to those previously described by Halie *et al.* in the blood of patients with Hodgkin's disease. Whereas none of the non-lymphoma patients had these cells, they were present in four of the five non-Hodgkin's lymphoma patients studied. Nineteen of 25 patients with Hodgkin's lymphoma had Halie cells, the presence of which appeared to correlate with disease activity. Two patients examined before and after splenectomy showed no change in the number of abnormal cells present. Lack of staging laparotomies in many patients precluded a statement regarding the previously proposed association between the presence of Halie cells and splenic involvement in Hodgkin's disease.

- 4605 SOME ASPECTS OF ION MOVEMENTS ACROSS THE MEMBRANE OF MITOCHONDRIA ISOLATED FROM NORMAL AND TUMOUR TISSUES. (E.) Rossi, C. S. (Dept. Med. Biochem., U. Nairobi, Kenya). *Afr J Med Sci* 4(3):293-299, 1973.

Calcium accumulation was studied in mitochondria

isolated from normal rat liver and in mitochondria isolated from Albino mice ascites tumor cells. Of 400 nmoles calcium added, tumor cell mitochondria accumulated 180 nmoles compared with 382 nmoles for normal mitochondria. The rate of calcium-stimulated respiration was 90 natoms/min/mg in normal mitochondria and 32 natoms/min/mg in tumor mitochondria. These data suggest that calcium movement across the mitochondrial membrane is a passive process in tumor cells. However, the translocation of the ion in the presence of the uncoupling agent dinitrophenol indicates that energy is required for the process. It is well known that transmission of a nerve impulse depends on a change in membrane permeability, due to liberation of the chemical transmitter. The phenomenon occurs at the level of the so-called chemical synapses, and modification of the permeability is accompanied by a conformational change in the postsynaptic membrane, resulting in an opening cation-specific channel.

- 4606 COMPETITIVE DNA-RNA HYBRIDIZATION OF MICROSOMAL AND NUCLEAR RNA IN NORMAL TISSUES OF THE RAT. (E.) Garrett, C. T. (U. Wisconsin Med. Sch., Madison), R. E. Moore, C. Katz and H. C. Pitot. *Cancer Res* 33(10):2464-2468, 1973.

Purified nuclear and microsomal RNA from rat brain, spleen, kidney, and liver were compared by means of competitive DNA-RNA hybridization. A close similarity in the sequences detected by this technique is noted in the nuclear RNA of kidney, brain, and liver, while a portion of these sequences appear to be missing from the nuclear RNA of spleen. Microsomal RNA from each of the four tissues appears to be qualitatively different. The precise role of reiterated sequences in DNA and RNA is as yet unknown. The finding of considerably greater sequence variability in microsomal RNA than in nuclear RNA would be consistent with a role for these sequences in the regulation of transport of microsomal RNA from nucleus to cytoplasm or in microsomal RNA template stabilization in the cytoplasm.

- 4607 FREEZE-ETCHING, SCANNING, AND THIN-SECTION ELECTRON MICROSCOPIC STUDIES OF THE "HAIRY" LEUKOCYTES IN LEUKEMIC RETICULO-ENDOTHELIOSIS. (E.) Burns, C. P. (U. Iowa Coll. Med., Iowa City) and J. C. Hoak. *J Natl Cancer Inst* 51(3):743-750, 1973.

Three techniques-freeze-etching, scanning, and thin-section electron microscopy-were used to study the ultrastructure of abnormal mononuclear leukocytes from three patients with leukemic reticulo-endotheliosis. Freeze-etching minimized artifactual distortion of the cytoplasmic "hairs," while allowing a three-dimensional-like view of the cell cleaved through membrane planes. Cleavage planes revealed no membrane abnormalities. Multishaped, tentacle-like projections of the cytoplasm contained few organelles. Cytoplasm contained mitochondria, endoplasmic reticulum, microfilaments, and granules. Nuclear pores were present in a high density similar to that in acti-

vated lymphocytes. Scanning electron microscopy, after critical point drying to decrease distortion, revealed abnormal cells with an extensive series of membrane outpouchings in these leukocytes but not in control preparations of cells from normal individuals and patients with chronic lymphocytic leukemia. Examination of thin sections revealed many cytoplasmic projections into redundant but apparently normal trilaminar membrane. Observation of these unique neoplastic cells by specialized ultrastructural techniques suggested that their major feature was the redundant membrane without unique morphologic membrane defects. The freeze-etch studies confirmed that the hairlike projections of the leukocytes were not artifacts.

- 4608 THE ARGININE CONTENT IN FIBRIN CLOTS AND FIBRINOLYSIS IN NEOPLASTIC DISEASES. (E.) Farbiszewski, R. (Med. Sch., Bialystok, Poland), W. Rzeczycki, K. Woroski and S. Glowinski. *Neoplasma* 20(2):203-208, 1973.

- 4609 REACTION OF THE SKELETON AND BONE MARROW TO MALIGNANT TUMOURS OF THE TESTIS. COMPARISON AND EVALUATION OF RESULTS OF STRONTIUM (⁸⁵Sr) TEST AND CYTOLOGICAL FINDINGS FROM STERNAL PUNCTATES IN 44 PATIENTS. (E.) Bek, V. (Sch. Gen. Med., Charles U., Prague, Czechoslovakia), Z. Hermanska, J. Kolar, J. Abrahamova and J. Jakoubkova. *Neoplasma* 20(2):209-215, 1973.

- 4610 MASSIVE PITUITARY ADENOMA IN A PATIENT WITH DYSTROPHIA MYOTONICA. (E.) Banna, M. (Newcastle U. Hosp., England), W. G. Bradley and G. W. Pearce. *J Neurol Sci* 20(1):1-6, 1973.

- 4611 THE ARGININE LEVEL IN GUERIN TUMOUR DURING ITS GROWTH IN RATS. (E.) Farbiszewski, R. (Med. Sch., Bialystok, Poland) and W. Rzeczycki. *Neoplasma* 20(2):217-219, 1973.

- 4612 HOST-TUMOUR RELATIONSHIP. XXXI. ACID GLYCOSAMINOGLYCANS IN THE PLASMA AND IN THE ASCITIC FLUID OF RATS DURING EXPERIMENTAL TUMOUR GROWTH. (E.) Skyvova, M. (Cancer Inst., Brno, Czechoslovakia), A. Kocent and I. Vermousek. *Neoplasma* 20(2):181-188, 1973.

- 4613 EVIDENCE AGAINST ASSOCIATION BETWEEN WET CERUMEN AND BREAST CANCER. (E.) Ing, R. (U. California, Sch. Med., San Francisco), N. L. Petrakis and H. C. Ho. *Lancet* (7793):41, 1973.

- 4614 GIANT FOLLICULAR LYMPHOMA OF THE STOMACH. REPORT OF 4 CASES. (E.) Akagi, T. (Kochi Prefectural Cancer Inst., Japan), K. Okajima, Y. Fujii and M. Ishikawa. *Acta Pathol Jap* 23(2):397-405, 1973.

- 4615 ULTRASTRUCTURE OF A TRANSPLANTABLE, MAMMO-SOMATOTROPIC PITUITARY TUMOR (MtT-W10). (E.) Nakayama, I. (Dept. Path. St. U. New York, Buffalo) and P. A. Nickerson. *Acta Pathol Jap* 23(2): 237-248, 1973.
- 4616 A STUDY ON THE INVASIVE GROWTH OF MALIGNANT TUMORS. II. ULTRASTRUCTURAL FEATURES OF THE METASTATIC GROWTH OF YOSHIDA ASCITES HEPATOMA 7974 IN THE RAT BRAIN. (E.) Machinami, R. (Fac. Med., U. Tokyo, Japan). *Acta Pathol Jap* 23(2):261-278, 1973.
- 4617 CHILDHOOD LEUKAEMIA PRESENTING IN THE CENTRAL NERVOUS SYSTEM. Graham Pole, J. (U. Dept. Child Hlth., Glasgow, Scotland). *Arch Dis Child* 48(11):867-871, 1973.
- 4618 CHROMOSOME STUDIES IN A THYMOMA *IN VITRO*. (E.) Kristoffersson, U. (Inst. Genetics, Lund, Sweden). *Humangenetik* 20(2):191-192, 1973.
- 4619 A METANEPHRINE TEST FOR A SCREENING OF CATECHOLAMINE-PRODUCING TUMORS. (E.) Sato, T. (Tohoku U. Sch. Med., Sendai, Japan) and K. Yoshinaga. *Tohoku J Exp Med* 110(3):283-287, 1973.
- 4620 MALIGNANT TUMORS IN MONKEYS. (E.) Kimbrough, R. D. (Bioeffects Branch, Environmental Protection Agency, Chamblee, Ga.). *Science* 181(4104):995, 1973.
- 4621 ULTRASTRUCTURAL FEATURES OF ALVEOLAR CARCINOMA OF THE LUNGS. (It.) Bucciarelli, E. (Inst. Pathol. Anat. Histol., U. Perugia, Italy). *Lav Ist Anat Istol Patol Perugia* 32(2):45-63, 1972.
- 4622 RETICULOENDOTHELIAL TUMORS IN MICE WITH TRANSPLANTS OF MAMMARY CARCINOMA ASCITES ATPC+. (It.) Maltzeff, N. (Cancer Res. Div., U. Perugia, Italy). *Lav Ist Anat Istol Patol Perugia* 32(2):65-83, 1972.
- 4623 CARCINOMA OF THE LARGE INTESTINE. (It.) Bolis, G. B. (Inst. Pathol. Anat. Histol., U. Perugia, Italy). *Lav Ist Anat Istol Patol Perugia* 32(2):85-93, 1972.
- 4624 RECURRENCES AND METASTASES OF CERVICAL CARCINOMAS. (Fr.) Dubois, J. B. (St. Eloi Hosp., Montpellier, France) and H. Pourquier. *Ann Radiol* 16(7/8):515-519, 1973.
- 4625 MALIGNANT LIVER TUMORS IN INFANTS. (Fr.) Gautier-Benoit, C. (Dr. Schaffner Hosp. Ctr., Lens, France), J. P. Cecile, M. Houcke, J. Lavielle, G. Verresse and H. Bayart. *Sem Hop Paris* 49(41):2685-2688, 1973.
- 4626 THE VALUE OF ROENTGENOGRAPHIC SCREENING (MAMMOGRAPHY) IN THE EARLY DIAGNOSIS OF BREAST CANCER. (Ger.) Dobretsberger, W. (Sisters of Mercy Hosp., Linz, Austria) and J. Kretz. *Oesterreich Z Erforsch Bekampf Krebskr* 28(3/4):73, 1973.
- 4627 RETICULUM CELL SARCOMA OF THE STOMACH WITH INVASION OF THE LIVER, SPLEEN AND BONE MARROW. (Fr.) van der Hoeden, R. (Hosp., Ixelles, Belgium) and R. Heimann. *Sem Hop Paris* 49(41):2675-2678, 1973.
- 4628 PRIMARY MULTIPLE CARCINOMAS OF THE GASTRO-INTESTINAL TRACT. (Ger.) Rosch, W. (Med. Clin., U. Erlangen-Nürnberg, Erlangen, Germany). *Dtsch Med Wochenschr* 98(40):1872-1873, 1973.
- 4629 PAINFUL DYSKERATOTIC TUMORS IN THE SUBINGUINEAL REGION. (Fr.) Pinol-Aguade, J. (Fac. Med., U. Barcelona, Spain), J. M. Mascaro, A. Castells-Rodellas, C. Herrero and C. Romaguera. *Bull Dermatol Syphiligr* 8(2):140-142, 1973.
- 4630 THE DIAGNOSTIC VALUE OF URINARY CYTOLOGY. (Ger.) Böck, D. (Munic. Hosp., Wien-Lainz, Austria) and G. Dobrovits. *Helv Chir Acta* 40(4): 545-547, 1973.
- 4631 HODGKIN'S DISEASE: ATTEMPT TO EVALUATE THE ROLE PLAYED BY APPENDECTOMY AND TONSILLECTOMY. (Fr.) Teillet, F. (St. Louis Hosp., Paris, France), C. Weisgerber and N. Feingold. *Nouv Presse Med* 2(32):2097-2099, 1973.
- 4632 MAMMARY CARCINOGENESIS: THE EQUIVOCAL ROLE OF PREGNANCY. (Fr.) Juret, P. (Francois Baclesse Ctr., Caen, France), J. Robillard, J. E. Couette, D. Brune and J. C. Vernhes. *Nouv Presse Med* 36(2):2412, 1973.
- 4633 RETROPERITONEAL MALIGNANT NEURINOMA. (Ger.) Wunderlich, M. (Altenburg District Hosp., Germany). *Zentralbl Gynaekol* 95(36):1291-1295, 1973.
- 4634 INTRAMUCOSAL CARCINOMA AND INVASIVE CARCINOMA OF THE STOMACH: NINE CASES. (Fr.) Marti, M. C. (Surg. Clin., U. Geneva, Switzerland), J. N. Cox, S. Widgren and C. Bouzakoura. *Schweiz Med Wochenschr* 103(42):1458-1462, 1973.
- 4635 SEVERE CORTISONE REACTIONS AFTER PROLONGED LOCAL CORTICOSTEROID THERAPY FOR MYCOSIS FUNGOIDES WITH XANTHOMATIZATION OF THE LESIONS. (Fr.) Rimbaud, P. (No affiliation), H. Serre, J. Meynadier and H. Baumelou. *Bull Dermatol Syphiligr* 8(2):176-178, 1973.

- 4636 ESOPHAGEAL DUPLICATION AND CANCER OF THE ESOPHAGUS. (Fr.) Luez, J. (Hosp. Ctr., Lens, France), B. Rossignol, F. Martin and P. Waghemacker. *Sem Hop Paris* 49(41):2657-2658, 1973.
- 4637 A LARGE, SLOW DEVELOPING (7 1/2 YEARS) CARCINOMA OF THE STOMACH WITH A NEGATIVE ENDOSCOPIC BIOPSY. (Fr.) Debray, C. (Bichat Hosp., Paris, France), J. Mendez, J. Pouliquen and C. Marche. *Sem Hop Paris* 49(41):2671-2673, 1973.
- 4638 CARCINOMA OF THE LARGE INTESTINE IN YOUNG ADULTS. (It.) Bolis, G. B. (Inst. Pathol. Anat. Histol., U. Perugia, Italy). *Lav Ist Anat Istol Patol Perugia* 32(3):103-108, 1972.
- 4639 CUTANEOUS AND MUCOSAL NEUROMAS WITH A HISTOPATHOLOGICAL AND ULTRASTRUCTURAL STUDY. (Fr.) Schnitzler, L. (Reg. Hosp., Angers, France), C. Simard, C. Daudoux and M. Lefranc. *Ann Dermatol Syphiligr* 100(3):241-260, 1973.
- 4640 PHENOTYPES OF THE REGAN ISOENZYME AND IDENTITY BETWEEN THE PLACENTAL D-VARIANT AND THE NAGAO ISOENZYME. (E.) Inglis, N. R. (Tufts U. Sch. Med., Boston, Mass.), S. Kirley, L. L. Stolbach and W. H. Fishman. *Cancer Res* 33(7):1657-1661, 1973.
- 4641 STRUCTURAL AND FUNCTIONAL CHANGES IN NOVICK-OFF HEPATOMA MITOCHONDRIA. (E.) White, M. T. (Dept. Molecular Biol., Biochem., U. California, Irvine) and K. K. Tewari. *Cancer Res* 33(7):1645-1653, 1973.
- 4642 *IN VITRO* LOCALIZATION OF ^{67}Ga IN EXFOLIATED EPITHELIAL CELLS FROM THE UTERINE CERVIX. (E.) Cobb, C. M. (American Sci., Engineering, Cambridge, Mass.), J. E. Zuckerman and M. Annis. *Cancer Res* 33(7):1578-1584, 1973.
- 4643 ADENOCARCINOMA OF THE BREAST IN WOMEN LESS THAN 30 YEARS OF AGE. (Fr.) Rouessé, J. (Gustave-Roussy Inst., Billejuif, France), G. Contesso, J. Génin, D. Sarrazin, J. Weiler and F. May-Levin. *Bull Cancer* 59(1):41-60, 1973.
- 4644 A SERUM SUBSTITUTE THAT CAN SUPPORT THE CONTINUOUS GROWTH OF MAMMARY TUMOR CELLS. (E.) Lasfargues, E. Y. (Inst. Med. Res., Camden, N.J.), W. G. Coutinho, J. C. Lasfargues and D. H. Moore. *In Vitro* 8(6):494-500, 1973.
- 4645 ANTITUMOR ACTIVITY OF *BACILLUS NATTO*. IV. PURIFICATION AND PROPERTIES OF AN EXTRACELLULAR PROTEASE FROM *BACILLUS NATTO* KMD 1126. (E.) Kameda, Y. (Fac. Pharmaceutical Sci., Kanazawa U., Japan), K. Matsui, K. Hosoya, A. Nomura and N. Sugano. *Chem Pharm Bull* 21(3):538-545, 1973.
- 4646 HODGKIN'S DISEASE IN CHILDHOOD. (E.) Young, R. C. (Natl. Insts. Health, Bethesda, Md.), V. T. DeVita and R. E. Johnson. *Blood* 42(2):163-174, 1973.
- 4647 PULMONARY BLASTOMA, REPORT OF A CASE. (E.) Vila, R. (Veterans Administration Hosp., Columbia, S. C.), J. J. McCoy, Jr. and R. E. McCall. *J SC Med Assoc* 69(7):251-256, 1973.
- 4648 CAVERNOUS HEMANGIOMA OF THE SPLEEN: ANGIOGRAPHIC OBSERVATIONS. (E.) Rosenthal, T. (Tel-Aviv U. Med. Sch., Tel-Hashomer, Israel), R. Adar, I. Wolfstein and V. Deutsch. *Angiology* 24(7):430-433, 1973.
- 4649 PLACENTAL AND FETAL INVOLVEMENT BY MATERNAL MALIGNANCY: A REPORT OF RECTAL CARCINOMA AND REVIEW OF THE LITERATURE. (E.) Rothman, L. A. (Mount Sinai Sch. Med. City U. New York), C. J. Cohen and J. Astarloa. *Am J Obstet Gynecol* 116(7):1023-1034, 1973.
- 4650 A MEDULLOBLASTOMA-LIKE TUMOUR WITH MELANIN FORMATION. (E.) Best, P. V. (Dept. Path., U. Aberdeen, Scotland). *J Pathol* 110(1):109-111, 1973.
- 4651 SPONTANEOUS THYMOMA IN BUFFALO RATS. (E.) Yamada, S. (Aichi Cancer Ctr. Res. Inst., Japan), K. Masuko, M. Ito and T. Nagayo. *Gann* 64(3):287-291, 1973.
- 4652 SPONTANEOUS DEVELOPMENT OF MACROPHAGE-LIKE CELLS IN A CULTURE OF MYELOID LEUKEMIA CELLS. (E.) Maeda, M. (Kyota U., Japan) and Y. Ichikawa. *Gann* 64(3):265-271, 1973.
- 4653 PRODUCTION OF GROWTH- AND DIFFERENTIATION STIMULATING FACTORS FOR MOUSE LEUKEMIA CELLS BY DIFFERENT CELL SPECIES. (E.) Maeda, M. (Kyota U., Japan) and Y. Ichikawa. *Gann* 64(3):257-263, 1973.
- 4654 MUCOPOLYSACCHARIDES OF RAT ASCITES HEPATOMA CELLS. (E.) Saito, S. (Sasaki Inst., Tokyo, Japan). *Gann* 64(3):247-255, 1973.
- 4655 INABILITY OF MURINE MELANOMA "TYROSINASE" (DOPA OXIDASE) TO OXIDIZE TYROSINE IN THE PRESENCE OR ABSENCE OF DOPA OR DIHYDROXYFUMARATE COFACTOR. (E.) Patel, R. P. (Tufts U. Sch. Med., Boston, Mass.), M. R. Okun, W. A. Yee, G. F. Wilgram and L. M. Edelstein. *J Invest Dermatol* 61(2):55-59, 1973.
- 4656 SOME CHARACTERISTICS OF THE PROLIFERATIVE ACTIVITY OF ERYTHROBLASTS IN UNTREATED AND TREATED ACUTE LEUKAEMIA. (E.) Queisser, W. (U. Ulm, Germany), A. Graubner, D. Hoelzer, U. Queisser and H. Heimpel. *Acta Haematol (Basel)* 49(5):271-280, 1973.
- 4657 ACUTE MYELOMONOCYTIC LEUKEMIA IN A PATIENT WITH HODGKIN'S DISEASE. (E.) Zwaan, F. E. (U. Med. Ctr., Leiden, Netherlands) and B. Speck. *Acta Haematol (Basel)* 49(5):291-299, 1973.

- 4658 BIOCHEMICAL STUDIES ON LIVER TUMORS OF CHILDREN. (E.) Murthy, A. S. K. (Children's Hosp. Med. Ctr., Boston, Mass.), G. F. Vawter, L. Kopito and E. Rossen. *Arch Pathol* 96(1):48-52, 1973.
- 4659 CONGENITAL MESOBLASTIC NEPHROMA AND ITS RECURRENCE. AN ULTRASTRUCTURAL OBSERVATION. (E.) Fu, Y.-S. (Med. Coll. Virginia, Richmond) and S. Kay. *Arch Pathol* 96(1):66-70, 1973.
- 4660 DNA DEPENDENT RNA POLYMERASE FROM EHRlich ASCITES TUMOR CELLS. II. FACTORS STIMULATING THE ACTIVITY OF RNA POLYMERASE II. (E.) Natori, S. (Fac. Pharmaceutical Sci., U. Tokyo, Japan), K. Takeuchi, K. Takahashi and D. Mizuno. *J Biochem* 73(4):879-888, 1973.
- 4661 HEREDITARY SACRAL AGENESIS ASSOCIATED WITH PRESACRAL TUMOURS. (E.) Kenefick, J. S. (Royal Free hosp., London, England). *Br J Surg* 60(4):271-274, 1973.
- 4662 BASAL CELL CARCINOMA IN CHILDREN. (E.) Milstone, E. B. (Armed Forces Inst. Path., Washington, D.C.) and E. B. Helwig. *Arch Dermatol* 108(4):523-527, 1973.
- 4663 FAMILIAL LYMPHOMA INCLUDING A REPORT OF FAMILIAL PRIMARY UPPER SMALL INTESTINAL LYMPHOMA. (E.) Banihashemi, A. (Dept. Med., Pahlavi U., Shiraz, Iran), K. Nasr, H. Hedayatee and H. Mortazavee. *Blut* 26(6):363-368, 1973.
- 4664 SELENIUM AND CANCER: CHEMICAL INTERPRETATION OF A PLASMA "CANCER TEST". (E.) Schrauzer, G. N. (U. California, San Diego), W. J. Rhead and G. A. Evans. *Bioinorg Chem* 2(7):329-340, 1973.
- 4665 ADENOSINE 3',5'-MONOPHOSPHATE IN CULTURED NEUROBLASTOMA CELLS: EFFECT OF ADENOSINE, PHOSPHODIESTERASE INHIBITORS AND BENZAZEPINES. (E.) Schultz, J. (Toxicology Inst., U. Tübingen, Germany) and B. Hamprecht. *Naunyn Schmiedeberg's Arch Pharmacol* 278(2):215-225, 1973.
- 4666 GASTRIC PSEUDOADENOMYOSIS IN A *MACACA MULATTA*. (E.) Andrews, E. J. (Pennsylvania State U. Coll. Med., Hershey) and W. J. White. *J Med Primatol* 2(1):19-24, 1973.
- 4667 INHIBITION OF ADRENOCORTICOTROPIC HORMONE PRODUCTION BY GLUCOCORTICOIDS IN MOUSE PITUITARY TUMOR CELLS. (E.) Watanabe, H. (Vanderbilt U. Sch. Med., Nashville, Tenn.), W. E. Nicholson and D. N. Orth. *Endocrinology* 93(2):411-416, 1973.
- 4668 STRUCTURAL ALTERATIONS OF SUPERMOLECULAR NUCLEOPROTEIN SYSTEMS INDUCED BY A DE-OXYRIBONUCLEOPROTEIN CONSTITUTENT OF TRANSFORMED MALIGNANT CELLS. (Rus.) Piritikhalaishvili, D. S. (Inst. Med. Genetics, Moscow, U.S.S.R.) and D. M. Spitkovskiy. *Biull Eksp Biol Med* 74(9):40-44, 1972.
- 4669 BLOOD BORNE METASTASIS IN CARCINOMA OF THE LARGE INTESTINE. (E.) Nahra, K. S. (No affiliation). *Leb Med J* 25(6):447-452, 1972.
- 4670 ON PRIMARY GASTROINTESTINAL LYMPHOMA IN IRAQ. (EXPERIENCE WITH 47 PATIENTS). (E.) Al-Bahrani, Z. E. (Med. City Hosp., Baghdad U., Iraq). *Leb Med J* 25(6):453-474, 1972.
- 4671 BASAL CELL EPITHELIOMA ARISING IN A DERM-ATOFIBROMA. (E.) Fishman, H. C. (No affiliation). *Cutis* 12(1):84-86, 1973.
- 4672 THE EFFECTS OF BIOPSY ON THE INCIDENCE OF METASTASES IN HAMSTERS BEARING MALIGNANT MELANOMA. (E.) Paslin, D. A. (U. Pennsylvania Sch. Med., Philadelphia). *J Invest Dermatol* 61(1):33-38, 1973.
- 4673 CHRONIC GRANULOCYTIC LEUKEMIA IN CHILDREN. (E.) Cooper, H. A. (Mayo Clinic, Rochester, Minnesota) and M. N. Silvestein. *Minn Med* 56(8):682-684, 1973.
- 4674 PURE GONADAL DYSGENESIS WITH BILATERAL GONADOBLASTOMAS. (E.) Farber, M. (Tufts U. Sch. Med., Boston, Mass.), P. E. Palmer and M. J. Bull. *Obstet Gynecol* 42(2):186-192, 1973.
- 4675 CYTOGENETIC STUDIES IN MYELOMA. (E.) Dartnall, J. A. (Dept. Med., U. Tasmania, Australia), G. R. Mundy and A. G. Baikie. *Blood* 42(2):229-239, 1973.
- 4676 DERIVATION AND BIOLOGIC PROPERTIES OF CELL LINES FROM OPHIDIAN TISSUES. (E.) Orr, H. C. (Cellular Physiology Branch, U.S. Dept. Hlth., Education, Welfare, Bethesda, Md.), P. G. Probst, J. L. Rogers, J. P. Davis, N. T. Stocks and J. Baker. *J Natl Cancer Inst* 51(3):827-832, 1973.
- 4677 SECOND CANCERS IN RETINOBLASTOMA. (E.) Strong, L. C. (U. Texas Hlth. Sci. Ctr., Houston) and A. G. Knudson, Jr. *Lancet* (7837):1086, 1973.
- 4678 CARCINOID TUMOURS OF THE COLON. (E.) Scott, J. E. (Princess Margaret Hosp., Swindon, England). *Br J Surg* 60(9):684-685, 1973.

- 4679 STATE OF THE CARDIO-VASCULAR SYSTEM IN PATIENTS WITH ADENOMA OF THE PROSTATE. (Rus.) Popov, A. I. (3rd Clin. Hosp., Zaporozhe, USSR). *Vrach Delo* 9:62-64, 1973.
- 4680 CHANGES OF SOME BILE COMPONENTS IN PATIENTS WITH CHRONIC LEUCOSES AND LYMPHOGRANULOMATOSIS. (Rus.) Sarnitsky, I. P. (Kiev Inst. Postgrad. Med., USSR) and V. T. Ishchenko. *Vrach Delo* 9:52-55, 1973.
- 4681 CHEMODECTOMAS OF THE LUNG. (Rus.) Badmaeva, V. V. (P. A. Gertsen Res. Inst. Oncol., Moscow) and I. Kh. Magomedova. *Arkh Patol* 35(1):45-49, 1973.
- 4682 THE STUDY OF NON-ORGANIC MESENCHYMATOUS TUMOURS OF THE HUMAN RETROPERITONEUM SPACE IN THE TISSUE CULTURE. (Rus.) Obukhova, L. E. (Inst. Exp. Clin. Oncol., Moscow, USSR) and G. K. Kusakina. *Arkh Patol* 35(1):31-38, 1973.
- 4683 CURRENT ASPECTS OF DIAGNOSTIC CYTOLOGY IN INVASIVE CERVICAL CARCINOMA. (Ger.) Bajardi, F. (Provincial Hosp., Graz, Austria) and E. Holzer. *Wien Klin Wochenschr* 85(42-43):714-718, 1973.
- 4684 LINEAR INFLAMMATORY WARTY EPIDERMAL NAEVUS (N.E.V.I.L.). LICHENOID DERMATOSIS OF NAEVIC TYPE, IN PLAQUES AND PRURIGINOUS BANDS, ITS RELATION WITH THE SOLOMON EPIDERMAL NAEVUS SYNDROME. (Fr.) Dupre, A. (Grave Hosp., Toulouse, France), B. Christol and M. L. Vialars. *Ann Dermatol Syphiligr* 100(3):261-274, 1973.
- 4685 SERUM ZINC CONCENTRATION: A UNRELIABLE PARAMETER FOR DIAGNOSING BRONCHOGENIC CARCINOMA. (E.) Smith, J. C., Jr. (VA Hosp., Washington, D.C.), H. H. Hansen, O. S. Selawry, M. P. Howard and J. A. Halsted. *J Natl Cancer Inst* 51(4):1379-1381, 1973.
- 4686 SPONTANEOUS PROSTATE ADENOCARCINOMAS IN AGED GERM-FREE WISTAR RATS. (E.) Pollard, M. (Lobund Lab., U. Notre Dame, Ind.). *J Natl Cancer Inst* 51(4):1235-1241, 1973.
- 4687 TRICHOBEZOAR, GASTRIC POLYPOSIS, PROTEIN-LOSING GASTROENTEROPATHY AND STEATORRHOEA. (E.) Hossenbocus, A. (Southampton Gen. Hosp., England) and D. G. Colin-Jones. *Gut* 14(9):730-732, 1973.
- 4688 DIFFERENTIAL UPTAKE OF $^{125}\text{I}^-$ AND $^{99\text{m}}\text{TcO}_4^-$ IN A HISTOLOGICALLY UNUSUAL METASTATIC THYROID CARCINOMA. (E.) Schall, G. L. (Natl. Insts. Health, Bethesda, Md.), J. A. Roth and R. Temple. *J Surg Oncol* 5(3):235-242, 1973.
- 4689 URINARY CONDUIT CYTOLOGY. (E.) Wolinska, W. H. (Mem. Hosp. Cancer, Allied Diseases, New York, N.Y.) and M. R. Melamed. *Cancer* 32(4):1000-1006, 1973.
- 4690 PHOSPHORYLATION OF SURFACE PROTEINS OF HELA CELLS USING AN EXOGENOUS PROTEIN KINASE AND $[\gamma\text{-}^{32}\text{P}]\text{ATP}$. (E.) Kinzel, V. (U. Wisconsin Med. Ctr., Madison) and G. C. Mueller. *Biochim Biophys Acta* 322(2):337-351, 1973.
- 4691 MESENCHYMAL CHONDROSARCOMA. A STUDY OF THE ULTRASTRUCTURE. (E.) Steiner, G. C. (Hosp. Joint Diseases, Med. Ctr., New York, N.Y.), J. M. Mirra and P. G. Bullough. *Cancer* 32(4):926-939, 1973.
- 4692 OVARIAN STROMAL TUMORS CONTAINING LEYDIG CELLS. I. STROMAL-LEYDIG CELL TUMOR AND NON-NEOPLASTIC TRANSFORMATION OF OVARIAN STROMA TO LEYDIG CELLS. (E.) Sternberg, W. H. (Tulane U. Sch. Med., New Orleans, La.) and L. M. Roth. *Cancer* 32(4):940-951, 1973.
- 4693 PRIMITIVE NEUROECTODERMAL TUMORS OF THE BRAIN IN CHILDREN. (It.) Hart, M. N. (Armed Forces Inst. Path., Washington, D.C.) and K. M. Earle. *Cancer* 32(4):890-897, 1973.
- 4694 BIOCHEMICAL AND HISTOLOGIC DETERMINANTS IN THE PROGNOSIS OF NEUROBLASTOMA. (E.) Gitlow, S. E. (Mount Sinai Sch. Med., City U. New York, N.Y.), L. B. Dziedzic, L. Strauss, S. M. Greenwood and S. W. Dziedzic. *Cancer* 32(4):898-905, 1973.
- 4695 THE SIGNIFICANCE OF PRIMITIVE CELLS IN MARROW ASPIRATES OF CHILDREN WITH NEUROBLASTOMA. (E.) Evans, A. E. (Children's Hosp., Philadelphia, Pa.) and K. Hummeler. *Cancer* 32(4):906-912, 1973.
- 4696 FIBROUS MASTOPATHY. A CLINICAL HISTOPATHOLOGIC STUDY. (E.) Minkowitz, S. (Dept. Surg., Path., St. U. New York, N.Y.), H. Hedayati, S. Hiller and B. Gardner. *Cancer* 32(4):913-916, 1973.
- 4697 ^{67}Ga SCINTIGRAPHY IN ACUTE LEUKEMIA. (E.) Milder, M. S. (Natl. Inst. Hlth., Bethesda, Md.), J. H. Glick, E. S. Henderson and G. S. Johnston. *Cancer* 32(4):803-808, 1973.
- 4698 PROLIFERATING HISTIOCYTIC LESION (HISTIOCYTOSIS-X?). ASSOCIATION OF AN EXTENSIVE MEDIASTINAL AND RETROPERITONEAL SCLEROSING LESION WITH GAGEL'S GRANULOMA OF THE POSTERIOR LOBE OF THE PITUITARY. (E.) Hou-Jensen, K. (Stanford U. Sch. Med., Calif.), D. G. Rawlinson and M. Hendrickson. *Cancer* 32(4):809-821, 1973.

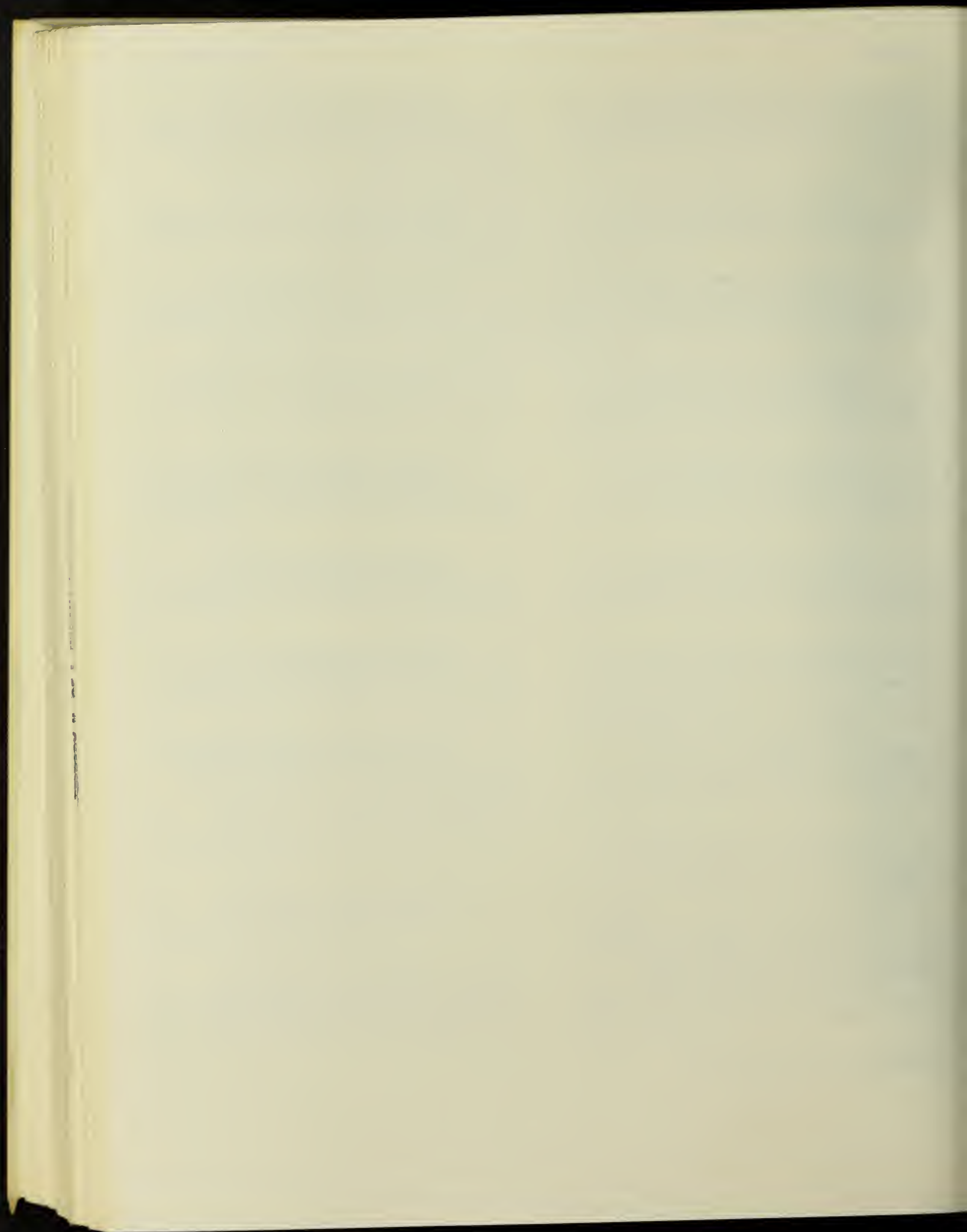
- 4699 IS THE DIFFERENTIATION BETWEEN PAPILLARY AND FOLLICULAR THYROID CARCINOMA VALID? (E.) Franssila, K. O. (Dept. Path., U. Helsinki, Finland). *Cancer* 32(4):853-864, 1973.
- 4700 ULTIMOBRANCHIAL THYROID NEOPLASMS IN BULLS. A SYNDROME RESEMBLING MEDULLARY THYROID CARCINOMA IN MAN. (E.) Black, H. E. (Dept. Vet. Pathobiol., Ohio St. U., Columbus), C. C. Capen and D. M. Young. *Cancer* 32(4):865-878, 1973.
- 4701 EVALUATION OF SERUM ALKALINE PHOSPHATASE DETERMINATION IN PATIENTS WITH POSITIVE BONE SCANS. (E.) Cowan, R. J. (Bowman Gray Sch. Med., Winston-Salem, N.C.) and K. A. Young. *Cancer* 887-889, 1973.
- 4702 ISOENZYMES OF LACTIC DEHYDROGENASE OF TUMOR TISSUE AND BLOOD SERUM IN PATIENTS WITH CANCER. (Rus.) Golubev, A. M. (Med. Inst., Provincial Oncological Dispensary, Astrakhanj, USSR), A. A. Chernukhin, L. I. Shvarts, L. D. Semenova, G. P. Mochalov and Ju. F. Palekhov. *Vopr Onkol* 19(9):46-50, 1973.
- 4703 AFFINITY OF $^{253}\text{EINSTEINIUM}$ FOR TUMOUR TISSUE. (E.) Hayes, R. L. (Oak Ridge Associated U., Med. Div., Tenn.), J. J. Rafter, L. C. Washburn and B. L. Byrd. *Nature [New Biol]* 246(149):23-25, 1973.
- 4704 CYCLIC GMP AND CELL MOVEMENT. (E.) Estensen, R. D. (Dept. Lab. Med., Path., U. Minnesota, Minneapolis), H. R. Hill, P. G. Quie, N. Hogan and N. D. Goldberg. *Nature [Lond]* 245(5426):458-460, 1973.
- 4705 IDENTIFICATION AND MISIDENTIFICATION OF THE CHROMOSOMES OF HETEROPOID CELL LINES. (E.) Walker, J. R. (U. Sheffield, Sub-Dept. Med. Genetics, England). *J Natl Cancer Inst* 51(4):1113-1117, 1973.
- 4706 EXTRA-ADRENAL PHEOCHROMOCYTOMA WITH METASTASIS IN DOWN'S SYNDROME. (E.) Kuni, C. C. (U. Colorado Med. Ctr., Denver). *J Pediatr* 83(5):835-836, 1973.
- 4707 CRANIOPHARYNGIOMA IN CHILDREN. (E.) Banna, M. (Regional Neurological Ctr., Newcastle-upon-Tyne U., England), R. D. Hoare, P. Stanley and K. Till. *J Pediatr* 83(5):781-785, 1973.
- 4708 HYPERPLASTIC AND METAPLASTIC RESPONSES OF HUMAN MAMMARY FIBROADENOMAS AND DYSPLASIAS IN ORGAN CULTURE. (E.) Elias, J. J. (U. California Med. Ctr., San Francisco) and R. C. Armstrong. *J Natl Cancer Inst* 51(4):1341-1343, 1973.
- 4709 MALIGNANT LYMPHOMA INVOLVING THE STOMACH. (E.) Kline, T. S. (Lankenau Hosp., Philadelphia, Pa.) and F. Goldstein. *Cancer* 32(4):961-968, 1973.
- 4710 PRIMARY MALIGNANT MESENCHYMAL TUMORS (MESENCHYMOMA) OF THE LIVER IN CHILDHOOD. AN ANGIOGRAPHIC-PATHOLOGIC STUDY OF THREE CASES. (E.) Stanley, R. J. (Mallinckrodt Inst. Radiology, St. Louis, Mo.), L. P. Dehner and A. E. Hesker. *Cancer* 32(4):973-984, 1973.
- 4711 LIGHT MICROSCOPIC IDENTIFICATION OF THE RIBOSOME-LAMELLA COMPLEX IN "HAIRY CELLS" OF LEUKEMIC RETICULOENDOTHELIOSIS. (E.) Katayama, I. (U. Hosp., Boston, Mass.), G. K. Nagy and K. Balogh, Jr. *Cancer* 32(4):843-846, 1973.
- 4712 STUDIES ON ECTOPIC ACTH-PRODUCING TUMORS: I. MEASUREMENT OF ACTH IN TUMOR TISSUE. (E.) Imura, H. (Kobe U. Sch. Med., Japan), S. Matsukura, H. Yamamoto, Y. Hirata, Y. Nakai and H. Matsuyama. *Jap J Clin Oncol* 6(3):7-12, 1973.
- 4713 HODGKIN'S DISEASE — A REPORT FROM BUENOS AIRES, ARGENTINA. (E.) Braylan, R. (J.M. Ramos Mejia Hosp., Buenos Aires, Argentina), H. Pascucci, M. Stadercker and M. C. Morgenfeld. *Cancer* 32(4):879-886, 1973.
- 4714 EXTRAMEDULLARY PLASMACYTOMATA OF THE UPPER RESPIRATORY TRACT. (E.) Booth, J. B. (Roy. Natl. Throat, Nose, Ear Hosp., London, England), A. D. Cheesman and N. H. Vincenti. *Ann Otol Rhinol Laryngol* 82(5):709-715, 1973.
- 4715 CONTROL OF INITIATION OF PROTEIN SYNTHESIS IN HUMAN CELLS. EVIDENCE FOR A ROLE OF UNCHARGED TRANSFER RIBONUCLEIC ACID. (E.) Vaughan, M. H. (Dept. Biochem., U. Pittsburgh, Pa.) and B. S. Hansen. *J Biol Chem* 248(20):7087-7096, 1973.
- 4716 GLUTAMATE-MEDIATED RESPIRATION IN TUMORS. (E.) Regan, D. H. (New York U. Med. Ctr., N.Y.), B. B. Lavietes, M. G. Regan, H. B. Demopoulos and H. P. Morris. *J Natl Cancer Inst* 51(3):1013-1017, 1973.
- 4717 TUMORS OF THE KIDNEYS, SYNOVIA, EXOCRINE PANCREAS, AND NASAL CAVITY IN BALB/CF/CD MICE. (E.) Rabstein, L. S. (Microbiol. Associates, Walkersville, Md.) and R. L. Peters. *J Natl Cancer Inst* 51(3):999-1006, 1973.
- 4718 DIFFERENTIATION OF NEUROBLASTOMA CELLS INDUCED IN CULTURE BY 6-THIOGUANINE. (E.) Prasad, K. N. (U. Colorado Med. Ctr., Denver). *Int J Cancer* 12(3):631-636, 1973.

- 4719 LEUKAEMIA-SPECIFIC D.N.A. AND TWINS. (E.) Knudson, A. G., Jr. (U. Texas, Hlth. Sci. Ctr., Houston). *Lancet* (7836):1032, 1973.
- 4720 CONGENITAL CYTOMEGALOVIRUS INFECTION AND BILEDUCT OBSTRUCTION IN NEWBORN INFANT WITH CYSTIC FIBROSIS OF PANCREAS. (E.) Oppenheimer, E. H. (Dept. Path., Johns Hopkins U. Baltimore, Md.) and J. R. Esterly. *Lancet* (7836):1031-1032, 1973.
- 4721 CONCURRENT MALIGNANCY IN T-CELL CHRONIC LYMPHOCYTIC LEUKAEMIA. (E.) Russell, J. M. (Western Infirm., Glasgow, Scotland) and A. J. Cochran. *Lancet* (7839):1215, 1973.
- 4722 ULTRASTRUCTURE OF CYTOPLASMIC HYALINE INCLUSIONS IN A CASE OF HUMAN HEPATOCARCINOMA. (E.) Enat, R. (VA West Side Hosp., Chicago, Ill.), R. J. Buschmann and B. Chomet. *Gastroenterology* 65(5):802-810, 1973.
- 4723 BREAST XERORADIOGRAPHY: AN ANALYSIS OF OUR FIRST 17 MONTHS. (E.) Frankl, G. (Southern California Permanente Med Ctr., Los Angeles) and D. D. Rosenfeld. *Ann Surg* 178(5):676-679, 1973.
- 4724 GASTRIC TERATOMA IN INFANCY: REPORT OF A CASE AND REVIEW OF WORLD LITERATURE. (E.) Matias, I. C. (William Beaumont Hosp., Royal Oak, Michigan) and Y. C. Huang. *Ann Surg* 178(5):631-636, 1973.
- 4725 RHINOPHYMA AND INTRANASAL CARCINOMA. (E.) Kornblut, A. D. (No affiliation) and K. Evers. *J Laryngol Otol* 87(11):1137-1141, 1973.
- 4726 METASTASES AND RECURRENCES IN NEPHROBLASTOMA. (E.) Jereb, B. (Karolinska Inst., Stockholm, Sweden). *Acta Radiol* 12(4):289-304, 1973.
- 4727 STUDIES ON THE TRANSFER OF INFORMATIONAL RIBONUCLEOPROTEIN COMPLEXES FROM NUCLEUS TO CYTOPLASM. (E.) Shapot, V. S. (Acad. Med. Sci., Moscow, U.S.S.R.) and A. V. Lichtenstein. *Neoplasma* 20(5):555-557, 1973.
- 4728 INTERACTIONS OF NORMAL AND NEOPLASTIC CELLS WITH VARIOUS SURFACES. (E.) Bershadsky, A. D. (Inst. Exp., Clin. Oncology, AMS, Moscow, U.S.S.R.), V. I. Guelstein, L. V. Domnina, O. Y. Ivanova, S. G. Komm, L. B. Margolis, Ju. M. Vasiliev and I. M. Gelfand. *Neoplasma* 20(5):583-585, 1973.
- 4729 MITOCHONDRIA IN ONCOGENESIS REVISITED. (E.) Schumacher, H. R. (Harrisburg Hosp., Pa.), I. E. Szekely and D. R. Fisher. *Lancet* (7839):1207-1208, 1973.
- 4730 A CONTAMINANT IN N-NITROSODIMETHYLAMINE CONFIRMATION BY HIGH RESOLUTION MASS SPECTROMETRY. (E.) Dooley, C. J. (U.S. Dept. Agriculture Eastern Regional Res. Ctr., Agricultural Res. Station, Philadelphia, Pa.), A. E. Wasserman and S. Osman. *J Food Sci* 38(6):1096, 1973.
- 4731 INSULIN AND ACTH PRODUCTION BY A STREPTOZOTOCIN RESPONSIVE ISLET CELL CARCINOMA. (E.) Walter, R. M. (U. Washington Sch. Med., Seattle), J. W. Ensink, H. Ricketts, J. W. Kendall and R. H. Williams. *Am J Med* 55(5):667-670, 1973.
- 4732 ENDOCRINE STUDIES IN AN ARRHENOBlastoma RESPONSIVE TO DEXAMETHASONE, ACTH AND HUMAN CHORIONIC GONADOTROPIN. (E.) Tucci, J. R. (Roger Williams Gen. Hosp., Providence, R.I.), W. Záh and A. E. Kalderon. *Am J Med* 55(5):687-694, 1973.
- 4733 A TRANSPLANTABLE MOUSE LYMPHOMA WITH UNUSUAL AZUROPHILIC GRANULES. (E.) Coppola, A. (Coll. Med., St. U. New York, N.Y.). *Am J Pathol* 73(1):233-246, 1973.
- 4734 PRELIMINARY HISTOLOGICAL AND ULTRASTRUCTURAL OBSERVATIONS ON A SPONTANEOUS TRANSPLANTABLE FIBROSARCOMA IN THE PERITONEUM OF THE GUINEA PIG. (It.) Bubola, G. (Inst. Histol. Gen. Embryol., U. Bologna, Italy), P. Pettazzoni and S. Biavati. *Boll Soc Ital Biol* 48(21):741-743, 1972.
- 4735 PRIMARY MEDIASTINAL TERATOCARCINOMA WITH GONADOTROPIN SECRETION. (E.) Ogura, T. (Osaka U. Hosp., Japan), T. Miyagawa, M. Ito, F. Hirao, Y. Yamamura and M. Hanada. *Jap J Clin Oncol* 6(3):53-62, 1973.
- 4736 SYSTEMIC CHLAMYDIAL INFECTION ASSOCIATED WITH GENERALIZED LYMPHEDEMA AND LYMPHANGIOSARCOMA. (E.) Elvin-Lewis, M. (Dept. Microbiol., Washington U. Sch. Dentistry, St. Louis, Mo.), M. Witte, C. Witte, W. Cole and J. Davis. *Lymphology* 6(3):113-121, 1973.
- 4737 HEMANGIOPERICYTOMA OF THE BRAIN. HISTOLOGICAL AND HISTOCHEMICAL STUDY OF FOUR CASES. (E.) Lolova, I. (Central Brain Res. Lab., Bulgarian Acad. Sci., Sofia) and M. Kamenova. *J Neurosurg* 39(5):636-641, 1973.
- 4738 HIDROCYSTOMAS. (E.) Smith, J. D. (Baylor Coll. Med., Houston, Tex.) and M. E. Chernosky. *Arch Dermatol* 108(5):676-679, 1973.
- 4739 DISSEMINATED HISTOPLASMOSIS AND ADENOCARCINOMA OF THE STOMACH. (E.) Kasch, J. A. (U. Kentucky Med. Ctr., Lexington) and U. W. Leavell, Jr. *Arch Dermatol* 108(5):698-699, 1973.

- 4740 RETROPERITONEAL LIPOBLASTIC TUMORS IN CHILDREN. (E.) Gonzales, E. T., Jr. (Duke U. Med. Ctr. (Duke U. Med. Ctr., Durham, N.C.) and E. E. Anderson. *J Urol* 110(4):474-475, 1973.
- 4741 CONGENITAL MESOBLASTIC NEPHROMA (LEIOMYOMATOUS HAMARTOMA): FIRST ADULT CASE. (E.) Block, N. J. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), H. G. Grabstald and M. R. Melamed. *J Urol* 110(4):380-383, 1973.
- 4742 PHEOCHROMOCYTOMA PRESENTING AS A URETEROCELE. (E.) Cabanas, V. Y. (De Paul Hosp., Norfolk, Va.), R. J. Faulconer and A. M. Fekete. *J Urol* 110(4):389-390, 1973.
- 4743 CARCINOMA OF THE PANCREAS CAUSING URETERAL OBSTRUCTION. (E.) Wanuck, S. (Beth Israel Hosp., New York, N.Y.), R. Schwimmer and L. Orkin. *J Urol* 110(4):395-396, 1973.
- 4744 GRANULAR CELL MYOBLASTOMA OF THE BLADDER: REPORT OF AN ADDITIONAL CASE. (E.) Mizutani, S. (Osaka U. Hosp., Japan), N. Okuda and T. Sonoda. *J Urol* 110(4):403-405, 1973.
- 4745 ULTRASTRUCTURAL DIFFERENTIATION OF PHEOCHROMOCYTOMA. (Ger.) Cervos-Navarro, J. (Neuropath. Inst., Free U. Berlin, Germany), J. M. Bayer and H. Käser. *Virchows Arch [Pathol Anat]* 361(1):51-69, 1973.
- 4746 FAMILIAL MULTIPLE POLYPOSIS. SURGERY CORE 1972. (E.) Hanna, R. W. (Med. U. South Carolina, Charleston). *J SC Med Assoc* 69(10):367-369, 1973.
- 4747 HERPES ZOSTER AND MULTIPLE MYELOMA. (E.) Saidi, P. (Rutgers Med. Sch., Camden, N.J.), W. E. Uhlman and I. Goldberg. *J Med Soc NJ* 70(11):836-838, 1973.
- 4748 GLOMUS TUMORS IN THE MIDDLE EAR. I. AN ANALYSIS OF 46 PATIENTS. (E.) Spector, G. J. (Washington U. Sch. Med., St. Louis, Mo.), R. H. Maisel and J. H. Ogura. *Laryngoscope* 83(10):1652-1672, 1973.
- 4749 PEDIATRIC HEAD AND NECK TUMORS: A STUDY OF 178 CASES. (E.) Jaffe, B. F. (Children's Hosp. Med. Ctr., Boston, Mass.). *Laryngoscope* 83(10):1644-1651, 1973.
- 4750 FIBROLIPOMA OF HYPOPHARYNX. CASE REPORT WITH 20 YEAR FOLLOW UP. (E.) McHenry, L. C. (No affiliation) and C. J. Wine. *J Okla St Med Assoc* 66(10):424-425, 1973.
- 4751 HEMANGIOMA-LIKE CLINICAL APPEARANCE OF A COLLAR-BUTTON MELANOMA CAUSED BY THE STRANGULATION EFFECT OF BRUCH'S MEMBRANE. (E.) Wolter, J. R. (U. Michigan Med. Ctr., Ann Arbor), A. L. Schut and C. L. Martonyi. *Am J Ophthalmol* 26(5):730-733, 1973.
- 4752 GLUTATHIONE PEROXIDASE IN HUMAN RED CELLS IN HEALTH AND DISEASE. (E.) Hopkins, J. (Dept. Pharmacology, Therap., U. Dundee, Scotland) and G. R. Tudhope. *Br J Haematol* 25(5):563-575, 1973.
- 4753 CYTOCHEMISTRY AND ULTRASTRUCTURE OF PATHOLOGIC GRANULATION IN MYELOGENOUS LEUKEMIA. (E.) Schmalzl, F. (U. Innsbruck Dept. Med., Austria), D. Huhn, H. Asamer, R. Rindler and H. Braunsteiner. *Blut* 27(4):243-260, 1973.
- 4754 MYXOMA OF THE MAXILLA — A CASE RECORD. (E.) Mostafa, H. M. (No affiliation), S. M. Abdellatif and A. Y. Montasser. *J Laryngol Otol* 87(11):1143-1146, 1973.
- 4755 MULTIPLE LEIOMYOMATA WITH ASSOCIATED CLITORAL HYPERTROPHY. (E.) Barricks, R. L. (U. Iowa Coll. Med., Iowa City). *J Iowa Med Soc* 63(11):535-538, 1973.
- 4756 INVASIVE PLEOMORPHIC ADENOMA OF HARD PALATE. (E.) Joachims, H. Z. (No affiliation) and M. M. Altman. *J Laryngol Otol* 87(11):1147-1151, 1973.
- 4757 TISSUE RECONSTITUTION WITH CULTURED HUMAN CANCER CELLS *IN VITRO* AND *IN VIVO*. (E.) Arata, T. (Okayama U. Med. Sch., Japan), I. Ogawa, Y. Nakazuma, Y. Tanaka and K. Sekiba. *Gann* 64(4):407-409, 1973.
- 4758 MEDIASTINAL TERATOMA WITH ENDOCRINE FUNCTION. (E.) Honicky, R. E. (U. Rochester Sch. Med., Dentistry, N.Y.) and E. W. dePapp. *Am J Dis Child* 126(5):650-653, 1973.
- 4759 CARCINOMATOUS OPTIC NEUROPATHY. (E.) Susac, J. O. (U. Miami Sch. Med., Fla.), J. L. Smith and J. O. Powell. *Am J Ophthalmol* 26(5):672-679, 1973.
- 4760 NEUROBLASTOMA. (E.) Truman, J. T. (Massachusetts Gen. Hosp., Boston). *Pediatrics* 1(4-5):231-238, 1972/1973.
- 4761 LUNG CANCER IN BOWEN'S DISEASE. (E.) Goldman, A. L. (Walter Reed Army Med. Ctr., Washington, D.C.). *Am Rev Respir Dis* 108(11):1205-1207, 1973.

- 4762 COEXISTING CEREBRAL AND INTRAMEDULLARY SPINAL GLIOMAS. (E.) Fountain, E. M. (Baylor Coll. Med., Houston, Tex.), M. S. Anderson and L. P. Pardo, Jr. *S Med J* 66(11):1306-1308, 1973.
- 4763 MESOTHELIOMA OF THE TUNICA VAGINALIS. (E.) Johnson, D. E. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), D. E. Fuerst and H. S. Gallager. *S Med J* 66(11):1295-1297, 1973.
- 4764 HYPERFUNCTIONING THYROID ADENOMA IN A EUTHYROID ADOLESCENT. (E.) Rosenbloom, A. L. (U. Florida Coll. Med., Gainesville) and M. M. Moore. *S Med J* 66(11):1247-1249, 1973.
- 4765 CAROTID BODY TUMORS: A 16-YEAR FOLLOW-UP OF SEVEN MALIGNANT CASES. (E.) Martin, C. E. (Vanderbilt U. Sch. Med., Nashville, Tenn.), L. Rosenfeld and B. McSwain. *S Med J* 66(11):1236-1243, 1973.
- 4766 CIRCADIAN RHYTHM IN THE SKIN TEMPERATURE OF NORMAL AND CANCEROUS BREASTS. PRELIMINARY REPORT. (E.) Mansfield, C. M. (Thomas Jefferson U. Hosp., Philadelphia, Pa.), R. A. Carabasi, W. Wells and K. Borman. *Int J Chronobiol* 1(3):235-243, 1973.
- 4767 TRACHEAL FIBROXANTHOMA IN A CHILD. (E.) Witwer, J. P. (U. Vermont Coll. Med., Burlington) and J. P. Tampas. *Postgrad Med* 54(5):228-229, 1973.
- 4768 CANCER DEVELOPMENT IN ORAL LICHEN PLANUS. A FOLLOW-UP STUDY OF 327 PATIENTS. (E.) Fulling, H.-J. (Roy. Dental Coll., Copenhagen, Denmark). *Arch Dermatol* 108(5):667-669, 1973.
- 4769 GIANT PROSTATIC CANCER WITH ACROMEGALY. (E.) Krawitt, L. N. (No affiliation). *J Urol* 110(4):441-442, 1973.
- 4770 BIOCHEMICAL INVESTIGATION OF CARCINOID TUMORS. (Fr.) Dreux, C. (St. Louis Hosp., Paris, France), B. Bousquet and D. Halter. *Ann Biol Clin* 31(4):283-294, 1973.
- 4771 THE EFFECTS OF LANTHANUM (La^{3+}) ON THE METABOLISM OF CALCIUM IN TUMORS. (E.) Anghileri, J. (Essen Clin., Ruhr U., Germany). *Int J Clin Pharmacol* 8(2):146-153, 1973.
- 4772 EXPRESSION OF LIVER PHENOTYPES IN CULTURED MOUSE HEPATOMA CELLS: SYNTHESIS AND SECRETION OF SERUM ALBUMIN. (E.) Bernhard, H. P. (Dept. Biol., Yale U., New Haven, Conn.), G. J. Darlington and F. H. Ruddle. *Dev Biol* 35(1):83-96, 1973.
- 4773 CONVERSION OF HYDROCORTISONE TO ESTROGEN IN CARCINOMA OF THE BREAST AFTER OOPHORECTOMY AND ADRENALECTOMY. (E.) Stonesifer, G. L., Jr. (Greater Baltimore Med. Ctr., Md.), R. H. Lowe, J. L. Cameron and F. M. Ganis. *Ann Surg* 178(5):563-564, 1973.
- 4774 GASTRIC LEIOMYOBlastoma: REPORT OF THREE CASES, ONE MALIGNANT. (E.) Abramson, D. J. (Washington Hosp. Ctr., Washington, D.C.). *Ann Surg* 178(5):625-630, 1973.
- 4775 SARCOMA BOTRYOIDES PRESENTING AS A POLYP ON THE LABIUM MAJUS. (E.) Talerman, A. (Inst. Radiotherap., Rotterdam, Netherlands). *Cancer* 32(4):994-999, 1973.
- 4776 CARCINOMA IN PLEOMORPHIC ADENOMAS OF SALIVARY GLANDS. (E.) Boles, R. (Dept. Otorhinolaryngology, Path., U. Michigan, Ann Arbor), M. E. Johns and J. G. Batsakis. *Ann Otol Rhinol Laryngol* 82(5):684-690, 1973.
- 4777 SEX CHROMATIN OF THE BUCCAL EPITHELIUM IN FEMALES WITH MALIGNANT NEOPLASMS. (Rus.) Voitenko, V. P. (U.S.S.R. Acad. Med. Sci., Moscow). *Vopr Onkol* 19(9):11-15, 1973.
- 4778 CONCERNING THE MORPHOLOGY AND CLINIC OF PARAGANGLIOMAS. (Rus.) Glezer, M. L. (Med. Stomatological Inst., Moscow, U.S.S.R.), O. B. Lisitsina and L. E. Bakhilov. *Vopr Onkol* 19(9):23-27, 1973.
- 4779 STUDIES ON MICRONUCLEOLI OF IMMATURE HUMAN LEUKEMIC NEUTROPHILS. (E.) Smetana, K. (Lab. Ultrastructure Res., Czechoslovak Acad. Sci., Prague), A. Vlastiborova and I. Iscenko. *Neoplasma* 20(5):491-498, 1973.
- 4780 THE EFFECTS OF INHIBITORS OF RNA AND DNA SYNTHESIS ON PROTEIN SYNTHESIS AND POLYSOME LEVELS IN MOUSE L-CELLS. (E.) Craig, N. (Dept. Biol. Sci., U. Maryland, Baltimore). *J Cell Physiol* 82(2):133-150, 1973.
- 4781 LYMPHOSARCOMA OF THE LUNG. (Fr.) Brocard, H. (Tenon Hosp., Paris, France), C. Choffel, H. Le Brigand, G. De Saint-Florent, P. Verdoux and G. Broquie. *Rev Tuberc Pneumol (Paris)* 36(4):541-556, 1972.
- 4782 PULMONARY HEMANGIOPERICYTOMA. REPORT OF 3 CASES. (Fr.) Uzzan, D. (Foch Hosp., Suresnes, France), J. Gilbert, J. Chebat, R. Asselain, L. Toty and C. Personne. *Rev Tuberc Pneumol (Paris)* 36(4):599-606, 1972.

- 4783 COLONIC LYMPHOMA IN ULCERATIVE COLITIS. A CASE REPORT. (E.) Parikh, N. K. (M.P. Shah Cancer Hosp., Ahmedabad, India), P. M. Shah, S. M. Patel and D. D. Chandra. *J Assoc Physicians India* 21(8):713-717, 1973.
- 4784 LYMPHOGRAPHY IN PATIENTS WITH TESTICULAR TUMOURS. (E.) Jonsson, K. (U. Hosp., Lund, Sweden), S. Ingemansson and L. Ling. *Br J Urol* 45(5):548-554, 1973.
- 4785 OBSERVATIONS ON URINARY CHEMILUMINESCENCE OF NORMAL SMOKERS AND NON-SMOKERS AND OF PATIENTS WITH BLADDER CANCER. (E.) Rose, G. A. (St. Peter's Hosp., London, England) and D. M. Wallace. *Br J Urol* 45(5):520-533, 1973.
- 4786 STUDIES ON SIALIC ACID IN YOSHIDA ASCITES SARCOMA CELLS. (E.) Rao, V. S. (Microbiol., Pharmacology Lab., Indian Inst. Sci., Bangalore) and M. Sirsi. *Indian J Biochem Biophys* 10(1):37-41, 1973.
- 4787 ARVIN-INDUCED HYPOFIBRINOGENEMIA AND METASTASIS FROM BLOOD-BORNE CANCER CELLS. (E.) Wood, S., Jr. (Merck Inst. Therap. Res., Rahway, N.J.) and P. H. Hilgard. *Johns Hopkins Med J* 133(4):207-213, 1973.
- 4788 PSEUDOSARCOMA OF THE ESOPHAGUS. A CASE REPORT. (E.) Sanchez, R. S. (St. John's Episcopal Hosp., New York, N.Y.), I. C. Shah, A. Barman, W. Batiuchok and J. E. Mule. *J Thorac Cardiovasc Surg* 66(5):833-837, 1973.
- 4789 GIANT CELL TUMOUR OF BONE. RADIOLOGICAL CHARACTERISTICS. (E.) Erens, A. C. (No affiliation). *Radiol Clin Biol* 42(5):385-394, 1973.
- 4790 DEMONSTRATION OF MALIGNANT TUMOURS IN THE LUNGS AND MEDIASTINUM BY MEANS OF RADIO-NUCLEAR (^{75}Se) SCINTIGRAPHY. (E.) Jereb, M. (Karolinska Hosp., Stockholm, Sweden), G. Unge, B. Jereb and G. Boman. *Scand J Respir Dis* 54(5):282-289, 1973.
- 4791 BONE TUMORS IN INFANTS AND CHILDREN. (E.) Nesbit, M. E., Jr. (Dept. Pediatrics, U. Minnesota, Minneapolis). *Paediatrician* 1(4-5):273-287, 1972/1973.
- 4792 SPINDLE CELL NEVI IN ADULTS AND CHILDREN. (E.) Coskey, R. J. (Wayne St. U. Sch. Med., Detroit, Mich.) and A. Mehregan. *Arch Dermatol* 108(4):535-536, 1973.
- 4793 NEVOID BASAL-CELL CARCINOMA SYNDROME. REPORT OF A PEDIGREE WITH ELECTRON MICROSCOPY OF SKIN LESIONS. (E.) Miller, A. S. (Temple U. Sch. Dentistry, Philadelphia, Pa.), C. Leifer, P. A. Pullon and M. W. Bowser. *Oral Surg* 36(4):533-543, 1973.
- 4794 FOCAL EPITHELIAL HYPERPLASIA IN AN ISRAELI FAMILY (E.) Buchner, A. (Fac. Continuing Med. Education, Tel Aviv U., Israel) and E. Mass. *Oral Surg* 36(4):507-511, 1973.
- 4795 CANCER OF THE HEAD AND NECK: EIGHT-YEAR EXPERIENCE. (E.) Berardi, R. S. (VA Hosp., Lexington, Ky.), F. Dominguez, R. L. Bradley and W. G. Malette. *S Med J* 66(10):1094-1100, 1973.
- 4796 ODONTOGENIC MYXOMA: ULTRASTRUCTURAL AND HISTOCHEMICAL STUDIES. (E.) Harrison, J. D. (King's Coll. Hosp. Dental Sch., London, England). *J Clin Pathol* 26:570-582, 1973.
- 4797 EFFECTS OF OBSERVER VARIATION IN POPULATION SCREENING FOR CERVICAL CARCINOMA. (E.) Lambourne, A. (Dept. Community Med., U. Sheffield, England) and H. Lederer. *J Clin Pathol* 26:564-569, 1973.
- 4798 THYROID STATUS AND BREAST CANCER. RE-APPRAISAL OF AN OLD RELATIONSHIP. (E.) Moossa, A. R. (U. Chicago Hosp., Clin., Ill.), D. A. Price Evans and A. C. Brewer. *Ann R Coll Surg Engl* 53(3):178-188, 1973.
- 4799 CHILDHOOD RHABDOMYOSARCOMA. (E.) Ragab, A. H. (St. Louis Children's Hosp., Washington U., Mo.). *Paediatrician* 1(4-5):288-297, 1972/1973.
- 4800 THE NATURE OF LIPOMAS AND THEIR SIGNIFICANCE IN THE ORAL CAVITY. A REVIEW AND REPORT OF CASES. (E.) Greer, R. O. (Boston U. Sch. Grad. Dentistry, Mass.) and J. F. Richardson. *Oral Surg* 36(4):551-557, 1973.



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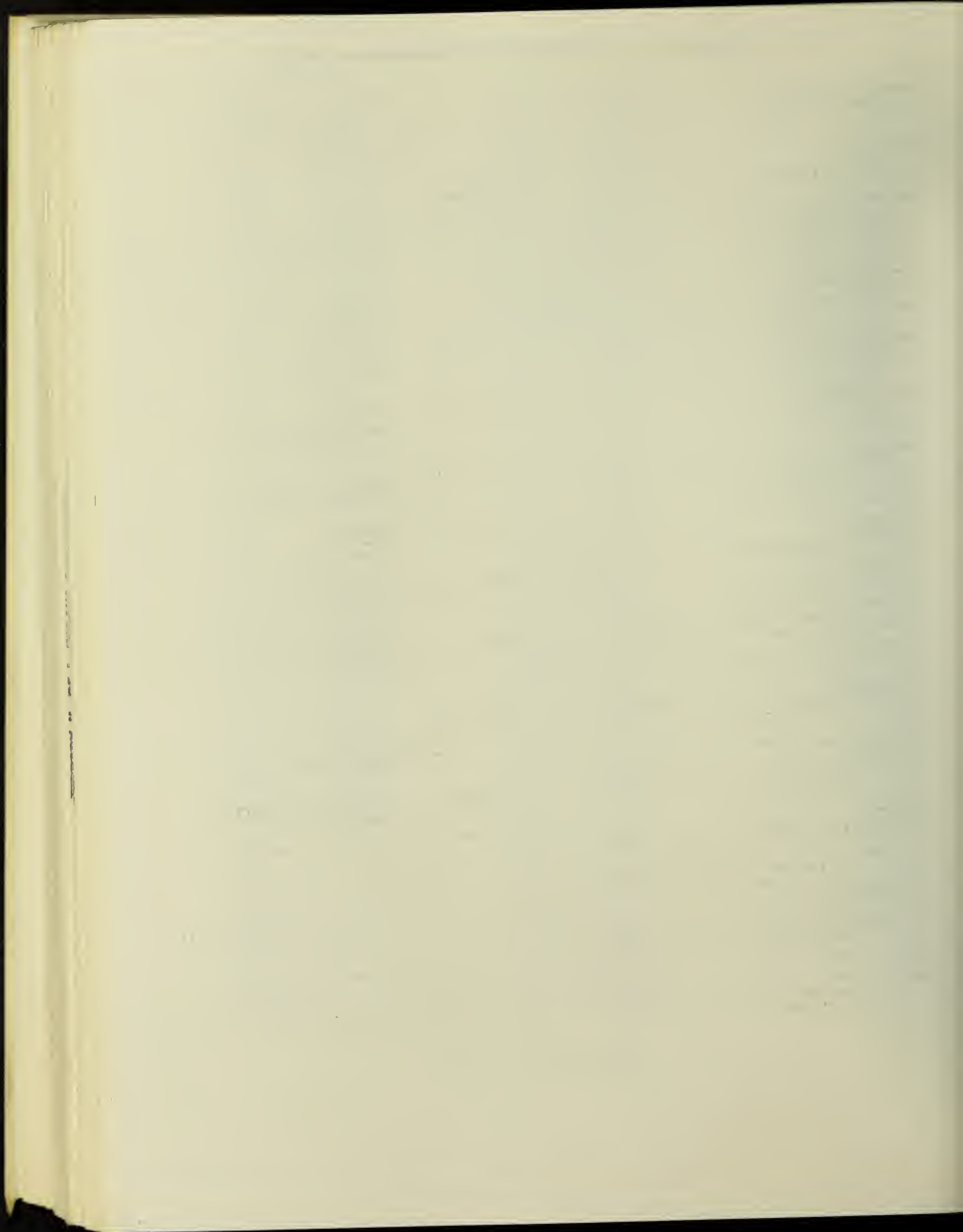
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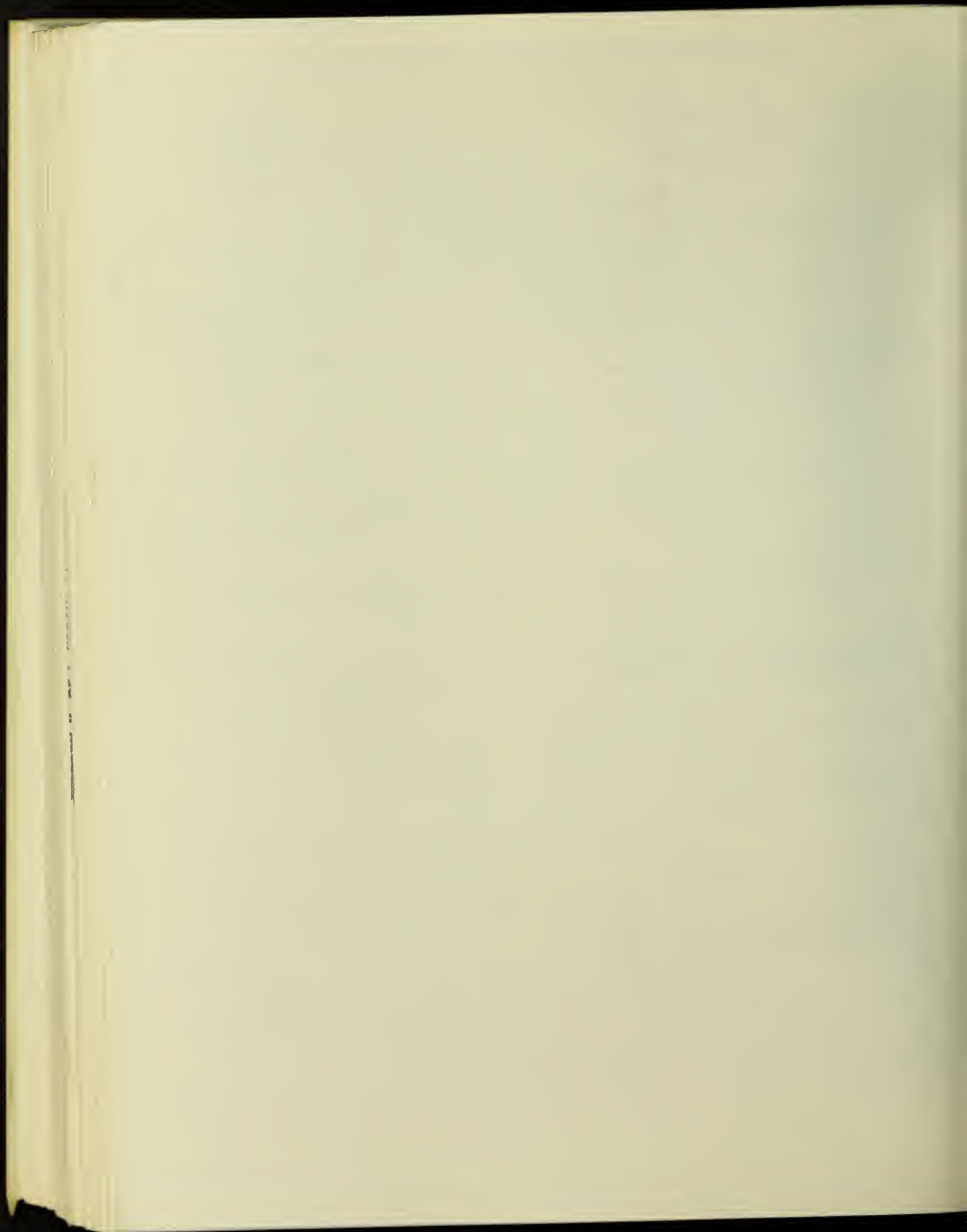
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CARCINOGENESIS ABSTRACTS

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CARCINOGENESIS ABSTRACTS

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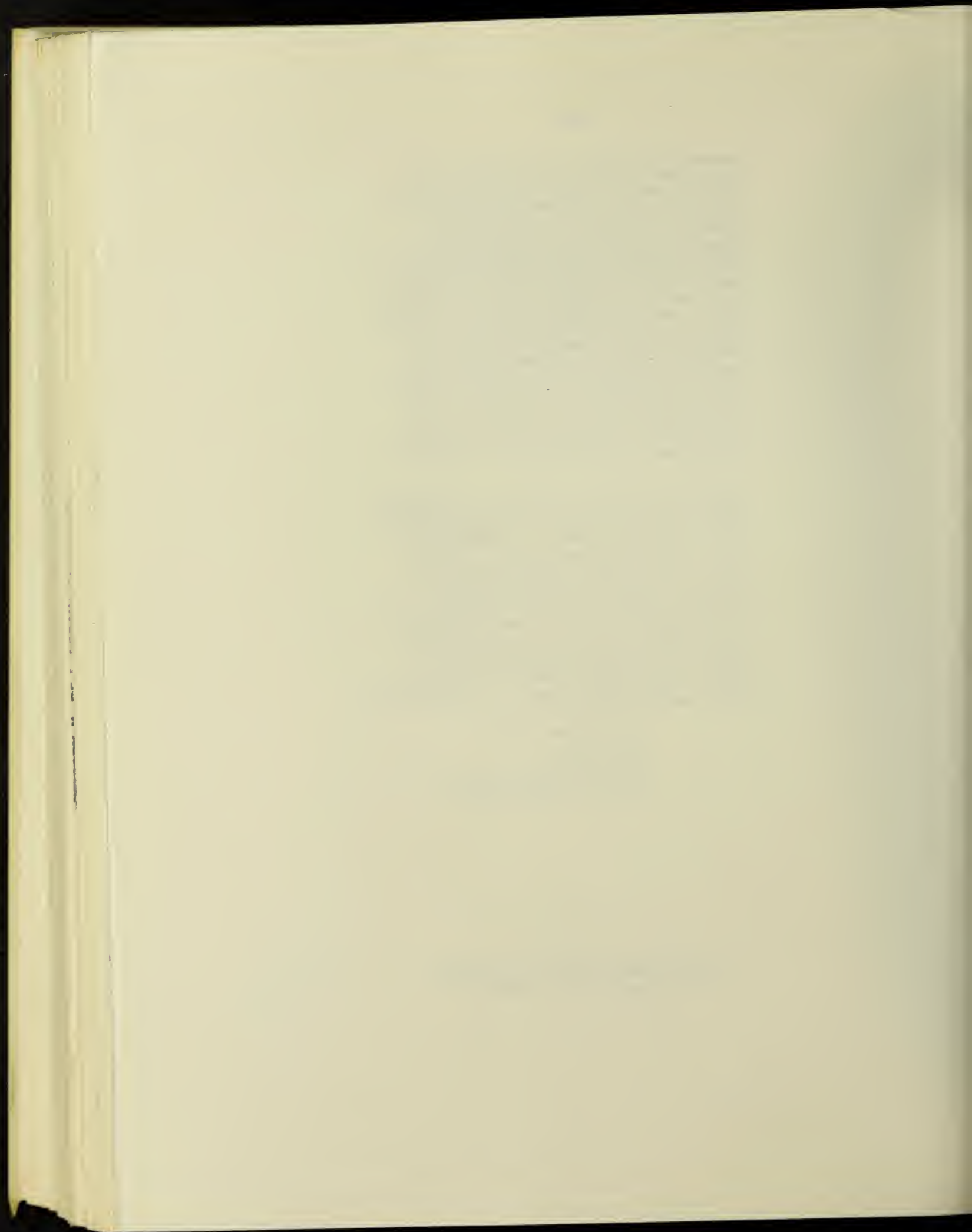
PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XI covers the scientific literature published from Jan 1973 through Dec 1973. A cumulative subject and author index for Volume XI will be published shortly after the final regular issue. The first issue of Volume XI which would normally be dated July 1972 is being dated July 1972 - January 1973. This change is being made so that the date of publication of material included in each issue corresponds to the issue date. This journal is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

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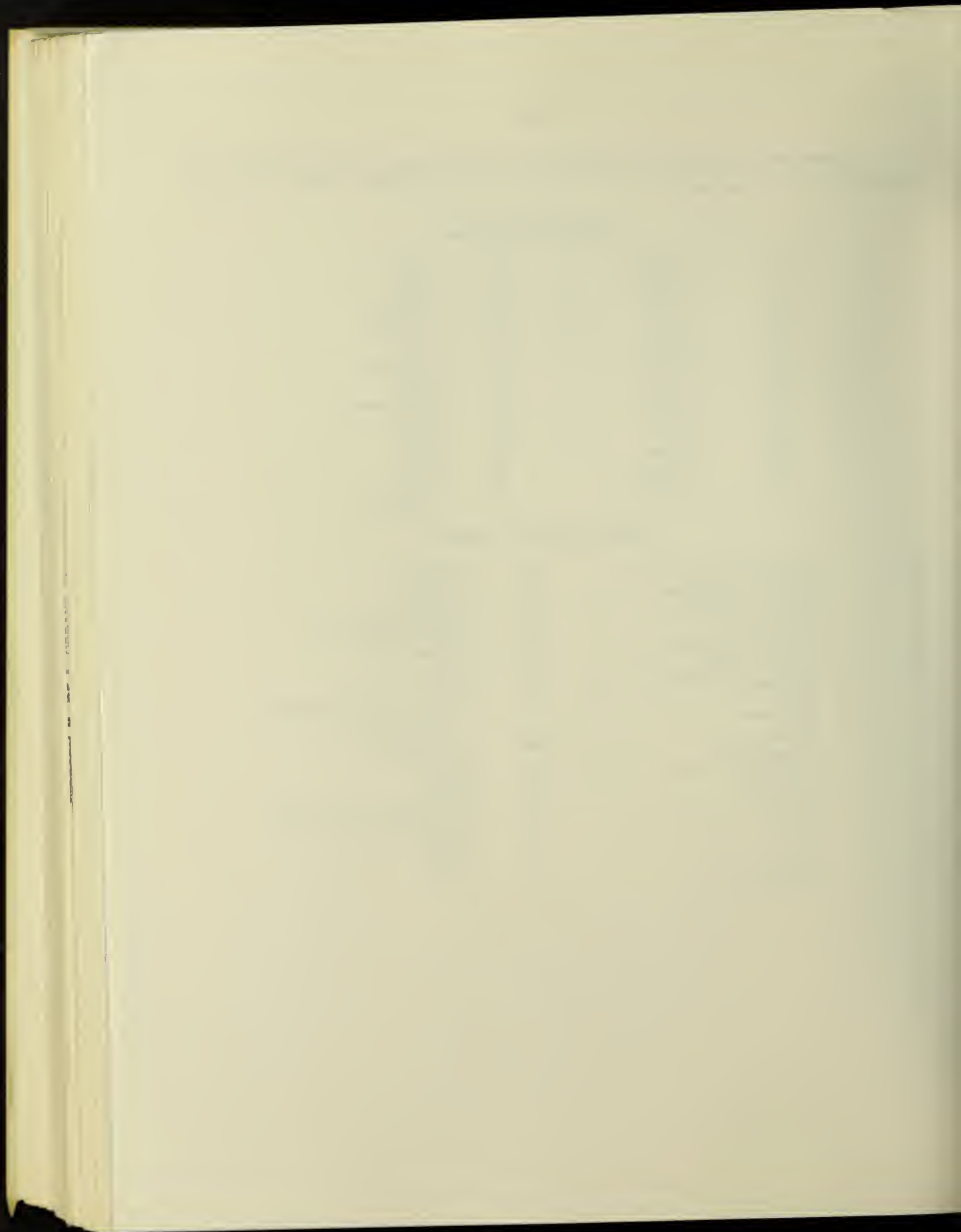
Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

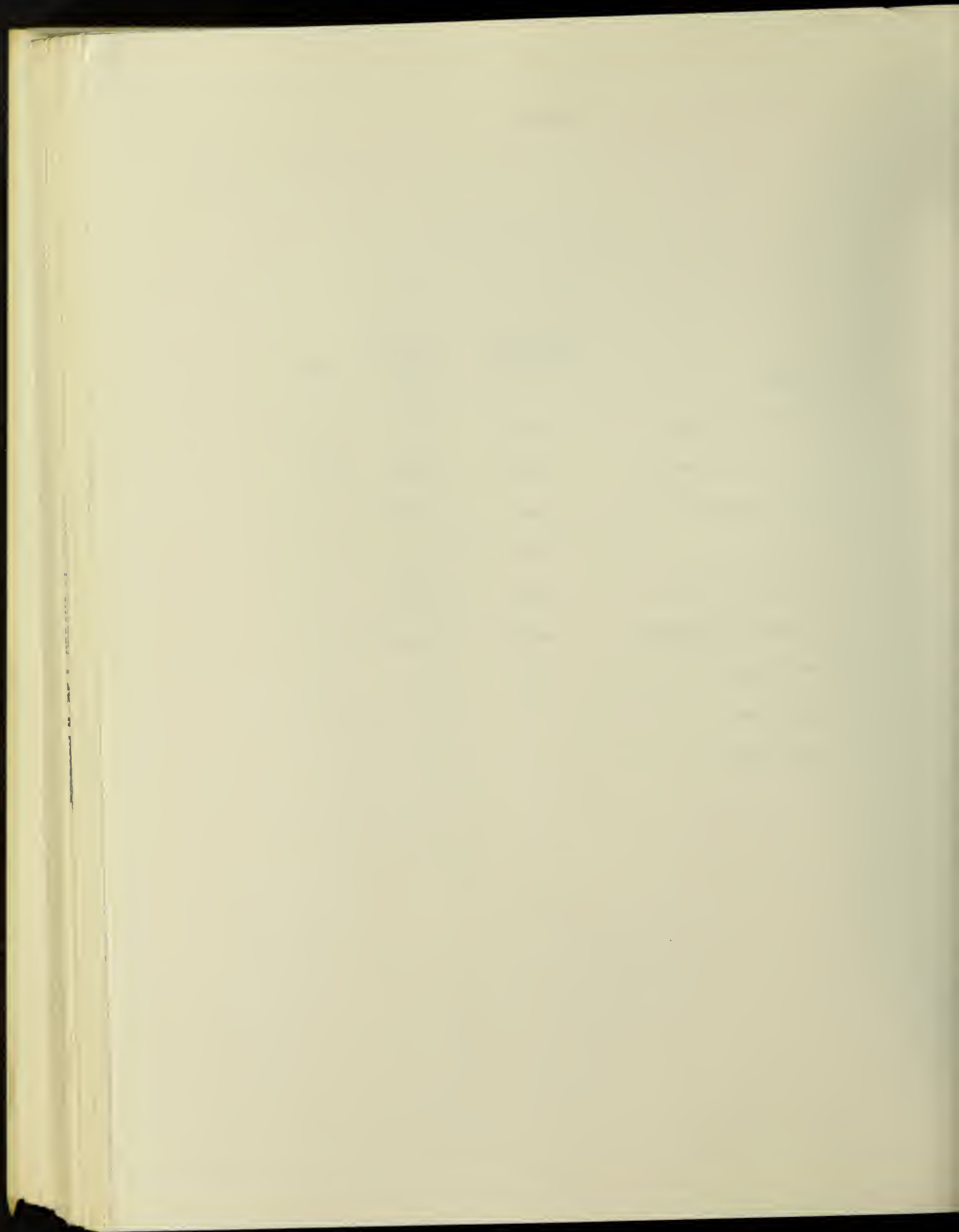
ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotrophic hormone	mg	milligram(s)
ADP	adenosine diphosphate	min	minute(s)
AMP	adenosine monophosphate	ml	milliliter(s)
ATP	adenosine triphosphate	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
µg	microgram(s)	resp.	respectively
hr	hour(s)	RNA	ribonucleic acid
i.m.	intramuscular	s.c.	subcutaneous
i.p.	intraperitoneal	sec	second(s)
IU	international unit(s)	U	unit(s)
i.v.	intravenous	UV	ultraviolet
kg	kilogram(s)	WBC	white blood cells (leukocytes), white blood count
LD ₅₀	median lethal dose(s)	wk	week(s)
m	meter(s)	wt	weight(s)
M	molar	yr	year(s)
mEq	milliequivalent(s)		
mM	millimolar		
µM	micromolar		
mC, µC	milli-, microcurie(s)		



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- 4801 NASOPHARYNGEAL FIBROMA: ITS HISTO-PATHOLOGICAL NATURE. (E.) Girgis, I. H. (Dept. Otolaryngol., Cairo U., Egypt) and S. A. Fahmy. *J Laryngol Otolology* 87(11):1107-1123, 1973.

Histological examination was made of 20 nasopharyngeal fibromas. Superficial undifferentiated epithelioid cells were found at the growing processes grouped in the form of cell nests, similar to those observed in glomus jugulare tumors. Some of the cells were large and vacuolated. The cells formed bigger collections in a deeper plane and inside these collections were blood spaces. Reticulin fibers were around the cell nests. Collagen fibers were present which rapidly matured into abundant fibrous tissue. These tumors showed no chromaffin reaction. Paraganglionic tissue was normally present in the terminal part of the maxillary artery and this may be the tissue of tumor origin. These results suggest that nasopharyngeal fibromas are abnormal growths of paraganglionic tissue resulting from endocrinal disturbance possibly occurring at puberty. The term "Glomus Nasopharyngis" is proposed as more accurate histopathologically. (36 references)

- 4802 STILBESTROL, ADENOSIS, AND ADENOCARCINOMA. (E.) Ulfelder, H. (Vincent Memorial Hosp., Boston, Mass.). *Am J Obstet Gynecol* 117(6):794-800, 1973.

A registry of cases of clear-cell adenocarcinoma of the genital tract in women under 30 yr of age was established in 1971. The first year's data amply confirmed the hypothesis that a simultaneous, nationwide outbreak of this lesion had occurred and that, in 85% of cases, exposure of the individual before the fourth month *in utero* to diethylstilbestrol or one of its congeners could be documented as an antecedent circumstance. General awareness of this new entity on the part of physicians and personal concern manifested by mothers who know that they were thus treated during pregnancy are bringing hundreds of the at-risk population group to medical attention. Gross and microscopic morphologic anomalies are being found in approximately 30% of these girls and occasionally asymptomatic early carcinomas are detected. A study of 16 patients who give a history of no prior exposure to hormones of any description reveals a difference between them and the group with known exposure. This is in keeping with the literature published prior to the availability of diethylstilbestrol which indicated that primary vaginal adenocarcinoma in young women was unique, whereas such lesions of the cervix were only uncommon. (18 references).

- 4803 SPECIFIC CARCINOGENIC AND TERATOGENIC EFFECTS OF 'INDIRECT' ALKYLATING METHYL AND ETHYL COMPOUNDS, AND THEIR DEPENDENCY ON STAGES OF ONTOGENIC DEVELOPMENTS. (E.) Druckrey, H. (Preventive Med. Res. Group, Freiberg, Germany). *Xenobiotica* 3(5):271-303, 1973.

The results of systematic studies on organospeci-

fic carcinogenesis and teratogenesis in which approximately 150 substances were comparatively tested in more than 10,000 BD rats are reviewed. Three groups of "indirect" carcinogens, dialkylnitrosamines, 1,2-dialkylhydrazines, azo- and azoxy-alkanes, and 1-aryl-3,3-dialkyltriazenes were investigated. Enzymic dealkylation and subsequent degradation of the resulting monoalkyl compounds eventually yielding the respective alkyl-diazonium as "proximate" alkylating carcinogen or teratogen, is a common feature of these three groups. In transplacental experiments, hydrazo, azo- and azoxy-ethane, as well as diethylaryltriazenes, proved highly carcinogenic to the fetus and induced malignant tumors exclusively of the brain and nervous system in almost all offspring even at a very low single dose. The sensitivity of the fetal nervous system to malignant transformation was demonstrable only after the 11th day. After administration of the ethyl-compounds at the 10th day, partial or complete inhibition of the development of the optic nerves and eyes, mostly associated with hydrocephalus internus, was observed with regularity. The dimethyl homologues and various dialkylnitrosamines were not carcinogenic to the fetus except at the last day of gestation. After birth all compounds revealed their respective organotropic effects. (77 references)

- 4804 CARCINOGENS IN PHARMACY AND THE ANALYTICAL CHEMISTRY LABORATORY. (Dut.) Reith, J. F. (Dept. Toxicol., U. Utrecht, Netherlands). *Pharm Weekbl* 107(37):581-597, 1972.

After a general discussion of chemical structure and carcinogenicity, species sensitivity, latent periods, differences in carcinogenic activity, factors involved in the carcinogenic action of a substance (cocarcinogens, anticarcinogens, solvents or vehicles, route of administration) and the dose-effect relationship, literature on the carcinogenicity of specific drugs and laboratory reagents is reviewed. The drugs considered include thorotrast, N,N-bis(2-chloroethyl)-2-naphthylamine, arsenic compounds, estrogens, isoniazide, thiourea derivatives, synthetic dyes (auramine, fuchsin, butter yellow, trypan blue, Evan's blue), urethane, macromolecular iron compounds, coal tar and petroleum products. Reagents used in analytical chemistry laboratories which are carcinogenic include benzidine, 2-naphthylamine, benzene, chloroform, carbon tetrachloride, nitrosophenols and diazomethane. Guidelines to be followed for the handling of carcinogenic aromatic amines are listed. (61 references)

- 4805 CANCER GENETICS, PART III: GENETIC MARKERS, CHILDHOOD CANCER PROBLEMS. (E.) Lynch, H. T. (Creighton U. Sch. Med., Omaha, Neb.). *Nebr Med J* 58(12):430-433, 1973.

The role of genetics and genetic markers in childhood cancers is briefly reviewed. It is suggested that childhood tumors may develop from a single, random cell abnormality. Once this abnormality occurs every descendant of that particular cell will

REVIEW

have the same chromosomal abnormality. Thus, chronic myelogenous leukemia (CML) was studied in a group of heterozygous patients, extracts of whose peripheral blood contained only a single enzyme type. This evidence favors a clonal origin of the leukemia cells in CML and supports the likelihood that the clone arises as a result of a rare event occurring in a single cell. Evidence exists in several autosomal recessive diseases associated with cancer in childhood showing that heterozygotes in the families of these children manifest an increased frequency of cancer. Heterozygous carriers of the gene for Fanconi's anemia, for example, show an increased frequency of cancer, and fibroblasts from these patients show increased transformation induced by SV-40 virus in tissue culture preparations. An example of several cases of cancer within a single family is given as an illustration of the genetic influences on childhood cancer development. (18 references)

- 4806 BOVINE LEUKEMIA: CURRENT STATUS OF THE PROBLEM. (Fr.) Parodi, A. L. (No affiliation). *Bull Mem Soc Med Paris* 176(6):41-45, 1973.

After a brief review of the clinical features of adult, juvenile and cutaneous forms of bovine leukemia, epidemiological evidence is presented which supports the hypothesis that the adult form of this disease is caused by a virus. The other two forms occur only sporadically. Since bovine leukemia affects all breeds of cattle, it is unlikely that genetic factors are involved in its transmission. However, many cases have been reported in which a mother and her offspring are affected, suggesting that there is a leukemogenic factor, possibly viral in nature, which is transmitted directly by the mother to the ovum or fetus or is ingested directly by the calf with colostrum or milk. Attempts to reproduce bovine leukemia by inoculating newborn calves or fetuses *in utero* with cell extracts from tumor cultures, blood, milk or colostrum have been successful in a few cases, but most animals only developed elevated WBC which is not a very specific criterion for the diagnosis of bovine leukemia. One investigator produced leukemia in 9 of 36 lambs inoculated s.c. with blood from a leukemic cow. Extra- and intra-cellular virus particles have been detected by the electron microscope in WBC cultures from leukemic cattle to which phytohemagglutinin or concanavalin had been added. This virus resembled type C particles of avian, murine and feline leukemias. Inoculation of these cultures into cattle increased the WBC in about 1/3 of the animals, and virus was recovered in lymphocyte cultures from all of the cattle. Virus was isolated from 60% of the cattle in a herd with a high incidence of bovine leukemia, but from only 10% in a herd with a low incidence of this disease. Specific precipitating antibodies were detected in 55% of the cases of leukemia occurring in adult cattle, but not in cattle with the cutaneous or juvenile forms of bovine leukemia. Antibodies have also been detected in the sera of sheep which developed leukemia after inoculation with blood or lymphocytes from leukemic cattle. (No references)

- 4807 SIGNIFICANCE OF IATROGENIC CARCINOGENESIS. (Ger.) Karrer, K. (Inst. Cancer Res., U. Vienna, Austria) and H. Wrba. *Verh Dtsch Ges Pathol* 56:164-168, 1972.

Although there is evidence that cytostatic agents are potentially carcinogenic, they are so effective against some forms of cancer, particularly acute leukemia, Burkitt's lymphoma and choriocarcinoma, that they should be used, even if the patients do develop reticulum cell sarcomas later. Palliative chemotherapy, combined with local radiotherapy, is also recommended in the treatment of inoperable solid tumors. Maintenance therapy is then advisable to prevent recurrences. Therapeutic nihilism is to be avoided at all costs, even by withholding information from the public about the alleged involvement of cytostatic agents in diaplacental carcinogenesis. (9 references)

- 4808 ACUTE CHILDHOOD LEUKEMIA AND BURKITT'S SARCOMA. ARE THEY ONLY RARE COMPLICATIONS OF COMMON INFECTIONS WHICH ARE GENERALLY NOTICED? (Fr.) Boyer, J. (Fac. Med., Paris, France). *Sem Hop Paris* 49(46):3085-3092, 1973.

Although it has been demonstrated that certain oncornaviruses induce leukemia and sarcomas in a variety of animal species, this has never been proven experimentally in man. In France, acute childhood leukemia occurs most frequently between the ages of 18 months and 5 yr. It is rarely diagnosed before the age of 6 months and is very rare after 15 yr. This suggests that the embryo or fetus could be infected by the mother or that acute leukemia is a rare complication of an undiagnosed infection. Maternal immunity could account for the rarity of acute leukemia in infants less than 6 months old, while its rarity in older children could be explained by an increase in acquired immunity to common infections. Burkitt's sarcoma is very rare in infants under 1 yr and in children older than 15 yr if they are natives of areas where this disease is endemic. The incidence of this tumor reaches a maximum in native children between 5 and 7 yr, but it can develop in foreigners several years after they immigrate to endemic areas. This suggests that the development of Burkitt's sarcoma is not related to age or race, but to receptivity to infection. These facts also rule out the possibility that the tumor is transmitted by the mother. If acute childhood leukemia and Burkitt's sarcoma are complications of common infections, their development could be affected by the number of virus particles and the virulence of the virus, but genetic factors could also play an important role. (No references)

- 4809 CLINICAL IMMUNOLOGY OF NEOPLASMS: CURRENT STATUS. (Pol.) Jasinski, W. (Inst. Oncol., Warsaw, Poland). *Pol Tyg Lek* 28(6):193-196, 1973. (14 references)

- 4810 SOME MECHANISMS INVOLVED IN LEUKEMIA CELL TRANSFORMATION. (Rus.) Butenko, Z. A. (Inst. Problems Oncol., Kiev, USSR). *Vest Akad Med Nauk SSSR* (4):74-78, 1973. (18 references)
- 4811 CHROMOSOME CHANGES AND CANCER. (Sw.) Mitelman, F. (Hosp., Lund, Sweden). *Lakartidningen* 70(16):1651-1654, 1973. (19 references)
- 4812 INTER-RELATIONSHIPS OF NUTRITION AND CANCER. (E.) Basu, T. K. (Marie Curie Mem. Fdn., Oxted, England), J. W. T. Dickerson and D. C. Williams. *Ecol Food Nutr* 2(3):193-199, 1973. (73 references)
- 4813 MALIGNANT LYMPHOMA - COMMENTS ON CURRENT STATUS. (E.) Greally, J. F. (Dept. Path., Trinity Coll., Dublin, Ireland). *J Ir Med Assoc* 65(9):229-232, 1972. (44 references)

- 4814 THE ACTION OF PROGESTIN ON THE ENDOMETRIUM AND TUMOR DEVELOPMENT IN THE CORPUS UTERI OF THE MOUSE AFTER IMPLANTATION OF 20-METHYLCHOLANTHRENE. (Ger.) Boquoi, E. (Clin. Obstet. Gynecol., Free U., Berlin, Germany) and H. Ebner. *Arch Gynaekol* 215(3):285-297, 1973.

A stylus containing 20-methylcholanthrene and beeswax was implanted into the uterus of 150 NMRI mice. Starting 6 days before carcinogen implantation, 50 of these mice received two doses of a progestin, 19-nor-17- α -hydroxyprogesterone caproate (200 μ g s.c. each), and another group of 50 mice received 200 μ g s.c. 6 days after implantation. Both of these groups continued to receive 200 μ g/wk of the progestin. After 22 wk significantly fewer uterine carcinomas had developed in progestin-treated mice than in controls. Although squamous cell metaplasia was common among the 100 progestin-treated mice, only 3 developed carcinoma *in situ*, 1 had a squamous cell carcinoma, and 2 had sarcomas. In contrast, 4 of the 50 controls developed carcinoma *in situ*, 2 had squamous cell carcinomas, 4 had mucoepidermoid carcinomas, and 4 had sarcomas. Progestin treatment 6 days before carcinogen implantation was particularly effective in slowing down tumor development. Tumors which did develop in these mice had slower rates of mitosis and were less mature than those found in the controls. Both of these phenomena are attributed to progestin treatment.

- 4815 THE MAMMOGRAM DURING CONTRACEPTIVE THERAPY. (Ger.) Brezina, K. (1st Clin. Obstet. Gynecol., U. Vienna, Austria), H. Janisch and E. Müller-Tyl. *Wien Klin Wochenschr* 85(48):785-790, 1973.

Mammographic examinations made on 480 women who were taking oral contraceptives indicate that oral contraceptives do not increase the incidence of breast cancer. Ethynylestradiol or mestranol were used as estrogens in combination with norethindrone, lynestrenol, norgestrel, or megestrol acetate or progestin were used alone. Mammograms taken before and during ingestion of oral contraceptives showed that mastopathies occurred in only 7 patients. Hyperplastic changes developed in 21 women and hypoplastic changes in 20. Hyperplastic changes were three times as common in multiparae as in nulliparae, while hypoplastic changes were four times as common in nulliparae as in multiparae. If the results of all 480 mammograms were analyzed, twice as many nulliparae (21.0%) as multiparae (12.2%) had mastopathies. One patient developed a carcinoma from fibroadenosis.

- 4816 5'-ENDONUCLEASE ACTIVITY IN THE LIVER DURING CARCINOGENESIS INDUCED BY 4-DIMETHYLAMINOAZOBENZENE AND INTRACELLULAR DISTRIBUTION OF THE ENZYME IN PRIMARY HEPATOMAS. (Fr.) Dupuis, C. (Notre Dame Hosp., Montreal, Canada) and R. Morais. *Rev Can Biol* 32(3):177-186, 1973.

Measurements were made of 5'-endonuclease in the nuclear, mitochondrial, lysosomal, microsomal and

soluble fractions of liver from male Wistar rats fed a protein-deficient diet and 0.06% 4-dimethylaminoazobenzene (DAB), from hepatomas induced with DAB and from untreated rats fed a protein-deficient diet. In controls and rats fed DAB for 2 wk, endonuclease was concentrated in the mitochondrial and nuclear fractions. In DAB-induced hepatomas, however, 19% of the activity was present in the nuclei, 26% in the mitochondria, and 22% in the lysosomal and soluble fractions, resp. Since cytochrome oxidase, and consequently mitochondria, contaminated the lysosomal fraction, most of the enzyme is present in the mitochondria. These values differ from those found for Novikoff's hepatoma where 68% of the endonuclease activity was present in the nuclear fraction, 7% in the mitochondria, and 4 and 26%, resp in the lysosomal and soluble fractions. The percentage of endonuclease in the soluble fraction of DAB-induced hepatomas was greater than that in controls but comparable to values found for Novikoff's hepatoma, suggesting that the enzyme may have escaped from the mitochondrial membranes which are very fragile in hepatomas. Mean activities of endonuclease were lower in rats fed DAB than in controls, but the difference was not significant. Endonuclease activities in hepatomas induced by DAB, however, were 15% lower than those found in controls. Since the mitochondrial fraction of tumor cells inhibits endonuclease activity in normal rat liver mitochondria, mitochondria in cancer cells may contain an enzyme inhibitor which would account for the decreased activity found in these organelles.

- 4817 THE EFFECT OF GONAECTOMY AND HORMONE ADMINISTRATION ON THE URINARY BLADDER CARCINOGENICITY OF N-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]FORMAMIDE IN MALE AND FEMALE SWISS MICE. (E.) Yoshida, O. (U. Wisconsin Med. Sch., Madison), E. Ertürk, G. T. Bryan and G. M. Lower, Jr. *Invest Urol* 11(3):216-220, 1973.

The urinary bladder carcinogen N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT) was fed to intact, gonadectomized, and gonadectomized (plus weekly s.c. injections of hormones) male and female Swiss mice at a dose of 0.05% by wt of diet for 22 wk, followed by 30 wk of control diet. No urinary bladder carcinomas occurred in male or female mice administered the control diet. In the groups of mice maintained on diets containing FANFT, bladder carcinomas occurred as follows: males - 3 out of 13 intact (23%), 4 out of 19 orchiectomized (21%), 5 out of 19 orchiectomized plus 1 μ g of 17 β -estradiol (26%), 6 out of 24 orchiectomized plus 10 μ g of 17 β -estradiol (24%), and 7 out of 20 orchiectomized plus 30 μ g of testosterone propionate (35%); females - 5 out of 18 intact (28%), 11 out of 19 ovariectomized (58%), 4 out of 18 ovariectomized plus 1 μ g of 17 β -estradiol (22%), 15 out of 24 ovariectomized plus 10 μ g of 17 β -estradiol (63%), and 2 out of 17 ovariectomized plus 30 μ g of testosterone propionate (12%). The data indicate a lack of sex difference in intact Swiss mice but suggest that the hormonal milieu alters susceptibility in female mice.

- 4818 PULMONARY CYTOLOGIC ALTERATIONS IN TOXIC ENVIRONMENTAL INHALATION. (E.) Frost, J. K. (Johns Hopkins U. Sch. Med., Baltimore, Md.), P. K. Gupta, Y. S. Erozan, D. Carter, D. H. Hollander, M. L. Levin and W. C. Ball, Jr. *Human Pathol* 4(4): 521-536, 1973.

Toxic elements in the environment adversely affect the tissues, structures and functions of the respiratory system. A number of defense mechanisms are operative in the respiratory tract to protect the host. A few of the key mechanisms can be effectively monitored by certain changes in the cells and other elements contained in the pulmonary secretions. These include the respiratory macrophages, the mucociliary blanket, and the trachio-bronchial epithelium. Specific changes which occur in these lines of defense as a result of damage by various environmental irritants are discussed. With regard to the collection of sputum, the most satisfactory specimen for the symptomatic ambulatory or hospitalized patient is the first produced in the morning upon awakening. These specimens are not adequate, however, for the asymptomatic and apparently healthy individual who is developing the epithelial atypias and other defects in the systems of respiratory defense. In such situations sputum induction aimed at sampling the entire bronchial tree is necessary. The techniques herein described were used to screen 1118 individuals in various risk industries. Both aerosol induced and spontaneous sputum samples were examined. The results indicated that the identification of atypical cells in aerosol induced pulmonary specimens appears to be a promising means of distinguishing an individual or an occupational group at high risk of developing lung cancer from one at low risk, even before actual cancers are uncovered.

- 4819 SUSCEPTIBILITY AND RESISTANCE TO ENVIRONMENTAL CARCINOGENS IN THE DEVELOPMENT OF CARCINOMA OF THE LUNG. (E.) Saccomanno, G. (St. Mary's VA Hosp., Grand Junction, Colo.), V. E. Archer, O. Auerbach and R. P. Sanders. *Human Pathol* 4(4):487-495, 1973.

Case histories of eight uranium miners, one of which worked in a uranium mill, are presented. Although seven of these men developed malignant tumors of the lung, each case presents a different clinical course in terms of the age of tumor development, the types of tumors, and the response to environmental exposure (amount of exposure and length of time of exposure). The known environmental carcinogens in this series were cigarette smoking and radon daughter exposure, singly and in combination. These exposures varied tremendously in amount and the patients showed a marked variation in response to them. It is concluded that the seven men who developed malignant tumors lacked the protective mechanisms against or resistance to these environmental carcinogenic exposures that are exhibited by some patients (e.g. the eighth case in this series, who was a heavy smoker and had 10 yr of hard-rock mining and 15 yr of uranium mining, but who developed no clinical or histologic evidence of disease). Some of the susceptible individuals seem to develop single or even multiple

cancers of the lung at a relatively young age or after a short latent period. Those individuals who develop malignant disease may have reduced immunologic or other responses.

- 4820 TOXIC, MUTAGENIC AND CARCINOGENIC EFFECTS OF ALKYLATING AGENTS. (Dut.) den Engelse, L. (Netherlands Cancer Inst.). *Chem Weekblad* 69(29):9-12, 1973.

The effect of mustard gas and other alkylating agents on DNA and the relationship of this effect to the toxic, mutagenic, teratogenic and carcinogenic activity of these compounds are reviewed. Because of their cytostatic action, which is most pronounced on rapidly growing cells, alkylating agents can be used in the treatment of cancer. The mutagenicity of alkylating agents is due to their direct interaction with DNA. If large quantities of β -propiolactone are painted on the skin of mice, no tumors develop, but tumors can be produced by applying a promoter such as bis(chloromethyl) ether or monochloromethyl-methyl ether. Alkyl mercury compounds present in the environment produce fetal malformations, and chlorodiphenyls can be carcinogenic. An increase in the incidence of tumors has been found in patients treated with immunosuppressants to prevent rejection of organ transplants. It has been demonstrated that hydrocarbons in the air can produce lung tumors in man, but it is likely that a number of other factors are also involved in the development of lung cancer. A comparison of the properties of N-methyl-N'-nitro-N-nitrosoguanidine and methylmethane sulfonate suggest a relation between the mutagenic and carcinogenic properties of these compounds.

- 4821 MESOTHELIOMA FOLLOWING EXPOSURE TO ASBESTOS: A REVIEW OF 72 CASES. (E.) Borow, M. (Somerset Hosp., Somerville, N.J.), A. Conston, L. Livornese and N. Schalet. *Chest* 64(5):641-646, 1973.

Out of 72 cases of mesothelioma arising apparently from occupational exposure to asbestos, 35 cases had histories of exposure ranging from 15-35 yr. Patients with the shortest exposure times included 2 males, aged 41 and 45 yr with less than 4 yr exposure each. In this series mesothelioma did not develop simultaneously in more than one serous cavity. Patients with pleural mesothelioma presented with mild chest pain or varying degrees of pleural effusion. A varying degree of pulmonary fibrosis was found. Initial diagnosis was made by needle pleural biopsy and Pap smear or by exploratory thoracotomy and biopsy. Peritoneal mesothelioma patients presented with vague abdominal discomfort, weight loss or distended abdomen. Diagnosis was made by peritoneoscopy and biopsy or by exploratory laparotomy. The growth remains on the surface and compresses either lungs or peritoneal cavity organs, but does not invade other organs. Histologically there are 2 types, and epithelial and the mesenchymal or fibrous type. It is suggested that the mesothelial tissue is the origin of these

tumors for the tumor cells produce significant quantities of mucopolysaccharides. Chemotherapy and radiation or combinations of modalities have not altered the course. Radiotherapy in combination with multichemotherapeutic agents as cyclophosphamide, nitrogen mustard, 5 fluorouracil actinomycin D or radioactive materials was not effective in this series. In the asbestos mill where these people were employed the main fiber used was chrysotile. It is suggested that factors in addition to asbestos acted as cocarcinogens; for example, only three of the above patients did not smoke.

- 4822 THE S-100 PROTEIN IN RAT BRAIN AND IN METHYLNITROSOUREA-INDUCED TUMORS OF THE RAT NERVOUS SYSTEM. A QUANTITATIVE STUDY. (E.) Stavrou, D. (Fac. Vet. Med., U. Munich, W. Germany), K. G. Haglid and L. Rönnebeck. *Eur Neurol* 10(3): 168-178, 1973.

S-100 protein levels were estimated in 29 of 48 nervous system tumors induced in Sprague-Dawley rats by twice weekly administration of methyl nitrosourea (MNU, 6 mg/kg) in drinking water. Tumors such as astrocytoma, oligodendroglioma, and mixed glioma did not differ significantly in water-soluble S-100 protein content from normal rat brain (mainly white matter). Polymorphous gliomas and neurinomas contained significantly lower amounts of water-soluble S-100 protein/mg soluble protein than did normal brain (mainly white matter). The presence of S-100 protein in 13 neurinomas of different peripheral nerves strongly supports a neuroectodermal origin for some MNU-induced neurinomas.

- 4823 CANCER AND STILBESTROL. A FOLLOW-UP OF 1,719 PERSONS EXPOSED TO ESTROGENS IN UTERO AND BORN 1943-1959. (E.) Lanier, A. R. (Mayo Grad. Sch. Med., Rochester, Min.), K. L. Noller, D. G. Decker, L. R. Elveback and L. T. Kurland. *Mayo Clin Proc* 48(11):793-799, 1973.

Follow-up studies were conducted on a group of 1,719 persons (901 males and 818 females) born between 1943 and 1959 who had been exposed to estrogens *in utero*. Of the 1,719, 57% had been exposed during the first trimester. In 93% of the cases, the estrogen used was synthetic diethylstilbestrol (Stilrone). Other drugs given included Premarin, ethisterone, progesterone, norethindrone, and hydroxyprogesterone. The dose of estrogen began at less than 5 mg/day and less than 4% of the mothers are known to have received a daily dose of 100 mg or more. The median age at follow-up was 22 yr for both males and females. No occurrence of invasion genitourinary cancer has as yet been reported and the incidence of cancer of other sites was not greater than that expected in the general population. Only 2 of the 126 deaths recorded have been attributed to malignancy: one with acute leukemia and the other with reticulum-cell sarcoma. Based on the administration of synthetic estrogens in the first trimester the risk of developing cancer of the vagina and cervix would be less than 7/1,000

by age 13 and less than 13/1000 by age 22 according to the results of this study. The combination of estrogens and progesterone in 25% of the cases receiving estrogens complicated conclusions concerning a link between estrogen and vaginal and cervical cancer.

- 4824 STUDIES ON CHEMICAL ALTERATIONS OF NUCLEIC ACIDS AND THEIR COMPONENTS. VI. N-AMINATION OF SOME NUCLEIC ACID BASES CONTAINING BASIC NITROGEN WITH HYDROXYLAMINE-O-ESTERS. (E.) Huang, G.-F. (Fac. Pharm. Sci., U. Tokyo, Japan), T. Okamoto, M. Maeda and Y. Kawazoe. *Tetrahedron Lett* 45:4541-4544, 1973.

N-amination of the basic nitrogens of cytidine, adenosine and related compounds was accomplished using hydroxylamine-O-sulfonic acid (HAOS) or 2,4-dinitrophenoxylamine (DNPA). Adenosine or cytidine was treated with 3-10 equivalent moles of HAOS in phosphate buffer at around a neutral pH at room temperature for several days. Only one product in each case was isolated in 10-40% yield. The use of DNPA and dimethylformamide afforded easier routes of preparation of 3-aminocytidine (from 1.0 mmole cytidine and 1.2 mmole of DNPA) and 1-aminoadenosine (from 10 mmole adenosine and 15 mmole of DNPA) instead of using HAOS and aqueous solvents. Related compounds which were studied were the triethylammonium salt of 3',5'-cyclic AMP (2.46 mmole with 3.96 mmole DNPA) and tubercidin (3.9 mmole with 4.5 mmole DNPA) which yielded the products 1-aminoadenosine-3',5'-cyclic monophosphate and 1-aminotubercidin, resp.

- 4825 SYMPOSIUM ON INDUSTRIAL CHEMICALS AS FOOD CONTAMINANTS. ASBESTOS FIBERS IN BEVERAGES, DRINKING WATER, AND TISSUES: THEIR PASSAGE THROUGH THE INTESTINAL WALL AND MOVEMENT THROUGH THE BODY. (E.) Cunningham, H. M. (Food Res. Labs., Dept. Natl. Hlth., Welfare, Ottawa, Ontario, Canada) and R. D. Pontefract. *J Assoc Off Anal Chem* 56(4):976-981, 1973.

Electron microscopic techniques were used to count the asbestos fibers present in various liquids and tissues. Fibers were detected in several samples of American and Canadian beer, sherry, port wines, vermouth, soft drinks, and city drinking water at levels ranging from 1.1 to 12.2 million fibers/liter. River water contained higher quantities of asbestos fibers than filtered city water and melted snow contained greater numbers than river water. In other experiments, chrysotile asbestos fibers (0.5-2 μ m in length) were injected into the stomachs of rats and samples of blood and tissues were analyzed for fibers after 2-4 days. Approximately 0.1% of the dose present in the blood after 2-4 days was detected in the tissues. Omentum surrounding the small intestine took up the largest amounts with brain and lung also showing high levels of fiber content. Experiments with 3 H-labeled and neutron-activated asbestos fibers indicated that spleen, liver, kidney, and muscle also contained variable amounts of fiber. Tissues of three humans who died of natural causes had levels of asbestos

fibers in brain, spleen, and peritoneum similar to those found in the experimental rats. The small amounts of asbestos fibers thus absorbed through the intestine may play an etiologic role in the development of certain visceral cancers.

- 4826 EFFECT OF SYNGENEIC BONE MARROW CELLS ON IMMUNOLOGICAL REACTIVITY AND CARCINOGENESIS IN THE LUNGS OF MICE TREATED WITH URETHANE. (Rus.) Kraskovskii, G. V. (Inst. Genetics Cytol., Acad. Sci. Belorussian SSR, USSR), L. S. Gorelik and L. F. Kagan. *Dokl Akad Nauk Beloruss SSR* 17(11):1052-1054, 1973.

In an effort to block the immunosuppressive and carcinogenic actions of urethane, 2-month-old male and female Af mice were given i.p. transplants of viable syngeneic bone marrow cells (15×10^6 or 30×10^6) or a mixture of 15×10^6 bone marrow cells and 35×10^6 spleen cells 24 hr after injection of a 10% urethane solution (1 mg/kg, i.p.). The immunological reactivity of these mice was evaluated by measuring hemolysis titers 4, 7, and 10 days after immunization with sheep RBC, and the number of pulmonary adenomas was counted 10 wk after administration of urethane. Urethane-treated mice who had received bone marrow or bone marrow and spleen cell transplants had higher hemolysis titers than urethane-treated controls. In urethane-treated mice both doses of bone marrow cells reduced the number of pulmonary adenomas by 40-53%, while the mixture of bone marrow and spleen cells reduced the number of these adenomas by 45-57%. Thus, transplantation of bone marrow or bone marrow and spleen cells protects mice from the carcinogenic and immunosuppressive actions of urethane.

- 4827 A NEW SYSTEM FOR QUANTITATIVELY EXPOSING LABORATORY ANIMALS BY DIRECT INHALATION. DELIVERY OF CIGARETTE SMOKE. (E.) Battista, S. P. (Life Sci. Div., Arthur D. Little, Inc., Cambridge, Mass.), M. R. Guerin, G. B. Gori and C. J. Kensler. *Arch Environ Health* 27(6):376-382, 1973.

A system for quantitatively introducing accurate volumes of cigarette smoke at specified frequency and duration into the respiratory tract of spontaneously breathing laboratory animals has been developed. The system has thus far been successfully used with chickens and dogs. Its use is currently restricted to animals either with tracheotomies or capable of mouth breathing. No anesthesia is required. The animal's respiratory activity does not determine, therefore, the volume frequency or duration of puffing. Smoke is loaded into a holding tube and inhaled during normal inhalation. To prevent anoxia, animals are allowed fresh air between puffs. The system can simultaneously expose 4-8 animals. Except for modest loss of particulate material, the composition of smoke does not appear altered materially. Possible disadvantages to this system include the aging of the smoke as it remains in the holding tube 2-10 sec., or the diluting of smoke for, as it reaches the lung, dilution may vary from one animal to another. It is suggested

that this system may prove useful in investigation of inhalation of other aerosols, fine dusts, and gases.

- 4828 EFFECT OF 3-AMINO-1,2,4-TRIAZOLE PRE-TREATMENT ON N- AND RING-HYDROXYLATION OF 2-ACETYLAMINOFLUORENE BY THE RAT. (E.) Lotlikar, P. D. (Temple U. Sch. Med., Philadelphia, Pa.), M. B. Wasserman and L. Luha. *Proc Soc Exp Biol Med* 144(2):445-449, 1973.

Adult Sprague-Dawley male rats were given aminotriazole (1g/kg/day, i.p.) for 9 days. At 4 hr after the last aminotriazole dose, an injection of 2-acetylaminofluorene (AAF) (30 mg/kg, i.p.) was received. A several-fold increase in the urinary excretion of N-hydroxy-2-acetylaminofluorene (N-hydroxy-AAF) was noted. The urinary excretion of ring-hydroxy metabolites of AAF was also appreciably increased. A similarly increased excretion of ring-hydroxy metabolites of AAF was achieved by bile-duct ligation of adult Sprague-Dawley male rats, with or without aminotriazole treatment. Ring-hydroxylation of AAF by liver microsomes was inhibited 25% in the aminotriazole treated group as compared to controls. Enzymatic esterification of N-hydroxy-AAF by liver cytosol preparations was inhibited by about 25% after the rats received aminotriazole (1g/kg/day) for 3 days. Such an aminotriazole effect may protect rats against liver carcinogenesis by AAF or the N-hydroxy derivative.

- 4829 TOXICOLOGICAL PROBLEMS OF IATROGENIC CARCINOGENESIS. (Ger.) Schmahl, D. (German Cancer Res. Ctr., Heidelberg). *Verh Dtsch Ges Pathol* 56:133-138, 1972.

It is recommended that long-term testing of drugs for carcinogenicity be restricted to: (1) agents suspected of being carcinogenic because of their constitution or chemical reactivity, e.g. alkylating agents and (2) agents given to pregnant women and children at relatively large doses over long periods. These agents should be administered to at least two different species of carcinogen-sensitive animals for the life span of the animal. The difficulties of extrapolating results of animal tests to man are considered. Although alkylating agents may be of great value in cancer chemotherapy, they should not be used in other conditions in which the immunosuppressive action of corticosteroids or antimetabolites would suffice. Problems involved in taking potential carcinogens off the market are illustrated by using phenacetin, diethylstilbesterol, and cyclamates as examples.

- 4830 MEASUREMENT OF ESTROGEN-BINDING CAPACITY IN THE HORMONE-DEPENDENT AND -INDEPENDENT RAT MAMMARY TUMORS. (E.) Nomura, Y. (Natl. Kyushu Cancer Ctr. Hosp., Japan), Y. Abe, T. Hattori, K. Inokuchi. *Cann* 64(4):401-404, 1973.

Mammary tumors were induced in female Sprague-Dawley

rats by intragastric administration of 7,12-dimethylbenz[a]anthracene. When a tumor measured at least 1.5 cm diameter, oophorectomy was performed. After oophorectomy, complete regression of the tumor was attained in 2 cases, and regrowth after a relatively long regression period was seen in a third. These tumors were hormone dependent. In 2 rats tumors recurred after a short regression. In 3 cases tumor regression was hardly noticeable and these tumors were regarded as hormone independent. Estrogen binding capacity of the tumors was measured. In hormone dependent tumor cytosols, very low concentrations of unlabeled estradiol competed with ^3H -estradiol for the binding sites, showing the binding to be highly specific. On the contrary, unlabeled estradiol showed little competition with ^3H -estradiol for the autonomous tumor cytosols. Therefore, high estradiol-binding capacities exist in the cytosols of tumors responding better to oophorectomy. Thus it is concluded that the hormone dependent mammary tumors retained some degree of the biochemical characteristics of normal estradiol target tissues, and the response to endocrine ablation therapy seems to be predicted by this method.

4831 THE CHEMISTRY OF NITROSO-COMPOUNDS. PART VI. DIRECT AND INDIRECT TRANSNITROSATION REACTIONS OF *N*-NITROSODIPHENYLAMINE. (E.) Challis, B. C. (Dept. Organic Chem., Imperial Coll., London, England) and M. R. Osborne. *J Chem Soc* (11):1526-1533, 1973.

The kinetics and mechanism of the interaction of *N*-nitrosodiphenylamine with various nucleophiles in aqueous solution were studied. Transfer of the nitroso-function from *N*-nitrosodiphenylamine to *N*-methylaniline, sodium azide, and other nucleophilic species is reported for acidic 50% aqueous ethanol at 25°C. Neutral *N*-nitrosodiphenylamine is unreactive and protonation is required to initiate these reactions. Transfer to *N*-methylaniline is not catalysed by added Cl^- , suggesting that the nitroso-group is transferred without the intermediacy of nitrous acid (*direct* transnitrosation). Transfer to sodium azide under similar conditions does proceed *via* nitrous acid. For other nucleophiles, however, both direct and indirect transnitrosation reactions may compete. Reaction rates are independent of these nucleophilic species when their concentration is high. Solvent isotope effects for reaction under these circumstances are negligible suggesting that an intramolecular rearrangement of the conjugate acid rather than protonation of the *N*-nitrosodiphenylamine is rate-limiting. Other nucleophilic species studied included hydroxylamine, sulphanilamide, *N*-1-naphthylethylenediamine, aniline, and 2-methylindole.

4832 OSTEOGENIC SARCOMA IN DIAL PAINTERS USING LUMINOUS PAINT. (E.) Martland, H. A. (No affiliation) and R. E. Humphries. *CA* 23(6):368-374, 1973.

Of 15 girls whose deaths were attributed to radium-mesothorium poisoning incurred while they were em-

ployed at painting watch dials with luminous paint, two were found to have osteogenic sarcoma of the bone. The mode of poisoning was by ingestion of small quantities of paint, small amounts of which were continually absorbed and stored as insoluble sulfates in the main organs of the reticulo-endothelial system and in the bones. After deposition in the bones, these deposits emitted their characteristic radiations day after day. Since about 95% of the radiation from these deposits was alpha, the blood forming centers were constantly bombarded by alpha particles. Thus, these centers eventually became exhausted, and leukopenic anemia developed, often proving fatal. One of the outstanding features of the early fatal cases was extensive, intractable necrosis of the jaw; while the later cases did not suffer from this problem, they did show chronic crippling lesions of the bones. The case history of one woman who died of radium mesothorium poisoning is presented. A diagnosis of osteogenic sarcoma of the scapula was made during life and proved at autopsy; the sarcoma developed following a fall in which the right shoulder was injured. It appears plausible that the sarcoma originated in a bone that had previously been the seat of a radiation osteitis, and that the preexisting deposits of radioactive substances in the bones played an important etiologic role in the subsequent development of the sarcoma.

4833 RELATIONSHIP BETWEEN TUMOUR CELL AND HOST IN CHEMICAL LEUKAEMOGENESIS. (E.) Haran-Ghera, H. (Weizmann Inst. Sci., Rehovot, Israel). *Nature [New Biol]* 246(151):84-86, 1973.

Female hybrid mice were exposed to 700 R whole body irradiation and were reconstituted within 1-3 hr with parental bone marrow of normal SJL/J mice or of SJL/J female mice previously treated for leukemia induction with 7,12-dimethylbenz[a]anthracene (DMBA, 4 weekly 1 mg feedings). Results indicated that no leukemias occurred in the irradiated hybrid mice reconstituted with normal parental SJL/J bone marrow cells, whereas the bone marrow from DMBA-treated hosts caused a 47.5% incidence of lymphatic leukemia at an average latent period of 205 days. Genotype analysis of 14 leukemias showed all were of donor origin. The presence of preleukemic or leukemic cells was established among transferred bone marrow cells collected from the mice treated with DMBA 7 days after termination of the carcinogen treatment. In experiments using SJL/J mice both as hosts and as bone marrow donors, the incidence of lymphatic leukemia from i.v. inoculation of bone marrow cells from DMBA treated mice was 90% at an average latent period of 145 days, whereas i.v. injection of normal isologous bone marrow cells did not cause leukemia. Within several days of termination of the leukemogenic treatment, proliferative preleukemic or leukemic cells were in the bone marrow, although the mice were clinically normal for some months. These results suggest that the transferred cells were not yet autonomous tumor cells, but were dependent on some further hyperplastic proliferation affected by host regulatory factors. Further work showed that host responses controlled the proliferation of tumor cells present in differ-

ent organs, and that occurrence of the disease is the end result of interaction between tumor cell and host, probably involving physiological regulatory processes and immunological responses.

- 4834 MORPHOLOGIC CHARACTERISTICS OF EXPERIMENTALLY INDUCED LUNG TUMORS AND THEIR PRECURSORS IN HAMSTERS. (E.) Stenbäck, F. (U. Nebraska Med. Ctr., Omaha). *Acta Cytol (Baltimore)* 17(6):476-486, 1973.

3-4 Benzopyrene in ferric oxide was administered intratracheally once weekly for 15 wk to Syrian golden hamsters. Each wk thereafter for 50 wk the lungs, tracheas, and mediastinal organs of two animals were examined. The neoplastic progression detectable in these organs during the course of the study was divided into three stages. The first was a reactive phase which was characterized by certain nonspecific alterations: ciliocytophthoria, slit formation and expulsion, basal cell hyperplasia, mucous metaplasia, squamous metaplasia, and squamous as well as adenomatous hyperplasia. In the pre-malignant phase, an adenoma, squamous papillomas, squamous dysplasia, and carcinoma *in situ* like changes were seen. The malignant phase was characterized by tumors which apparently originated from a common precursor differentiating into typical adeno and squamous cell carcinoma, a large cell carcinoma-like tumor occurring very late in the experiment. It is concluded that the development and progression of tumors in the respiratory system of the hamster is in many aspects similar to that seen in humans.

- 4835 CARCINOGENICITY OF TAR-CONTAINING SKIN DRUGS: ANIMAL EXPERIMENT AND CHEMICAL ANALYSIS. (E.) Hirohata, T. (Fac. Med., Kyushu U., Fukuoka, Japan), Y. Masuda, A. Horie and M. Kuratsune. *Gann* 64(4):323-330, 1973.

Tar-containing skin drugs, Pityrol, Glyteer, Ichthammol JP, pine tar JP, and Metashal, were investigated by animal experiments and by chemical analysis to determine their carcinogenicity. Animal experiments showed that the proportions of skin papilloma-bearing mice as compared to those at the first appearance of tumor were 64% (Pityrol), 71% (Glyteer), 9% (Ichthammol), 23% (pine tar), and 64% (Metashal). The proportions of skin carcinoma-bearing mice were 36% (Pityrol), 40% (Glyteer), 0% (Ichthammol), 6% (pine tar), and 22% (Metashal). A high proportion (37%) of metastasis in adjacent or remote organs was observed. No tumors developed in the control group receiving acetone only. A substantial amount of polycyclic aromatic hydrocarbons were found in the drugs, and, in particular, benzo[a]pyrene was identified, and its content agreed well with the degree of carcinogenic activity observed by the animal experiment. The average amount of benzo[a]pyrene, from three measurements, were 145 (Pityrol), 129 (Glyteer), none (Ichthammol), 48 (pine tar), and 80 µg/10g (Metashal). It is suggested that a long-range study of patients receiving these drugs be undertaken.

- 4836 EXPERIMENTAL PRODUCTION OF LINGUAL CARCINOMAS IN HAMSTERS: TUMOR CHARACTERISTICS AND SITE OF FORMATION. (E.) Fujita, K. (Sapporo Med. Coll., Japan), T. Kaku, M. Sasaki and T. Onoe. *J Dent Res* 52(6):1176-1185, 1973.

Lingual carcinomas were produced at various sites on the hamster tongue by local application of 9,10-dimethyl-1,2-benzanthracene (DMBA). Ninety-one Syrian golden male hamsters, two months old were used. A 0.5% DMBA-acetone solution was used as the carcinogen. The period of carcinogenesis, degree of infiltration, and frequency of metastases were different according to site. The period of carcinogenesis is relatively short in the lateral border of the middle third of the tongue, longer in the lateral border of the anterior third, the undersurface, and the tip of the tongue, and very long in the midportion of the dorsum of the tongue. Infiltration and metastases were most frequent in the tumors produced in the lateral border of the middle third of the tongue. The experimental results were similar to clinical manifestations of human lingual carcinomas, suggesting that this experimental lingual carcinoma is a reasonable model for human lingual carcinoma.

- 4837 TRANSFORMATION OF CELL CULTURES AS AN INDICATION OF THE CARCINOGENIC POTENTIAL OF CHEMICALS. (E.) Freeman, A. E. (Children's Hosp., Akron, Ohio), E. K. Weisburger, J. H. Weisburger, R. G. Wolford, J. M. Maryak and R. J. Huebner. *J Natl Cancer Inst* 51(3):799-807, 1973.

Over 30 polycyclic hydrocarbons, azo dyes, aromatic amines, and miscellaneous chemicals were tested to see if *in vitro* transformation of high-passage (Fischer) rat embryo cultures correlated with the known carcinogenic activity of the same chemicals in animals. In general, *in vitro* cell transformation was induced by known carcinogens but not by their noncarcinogenic analogues. The results with most compounds were consistent from experiment to experiment. Anthracene, fluoranthene, and phenanthrene were among the exceptions. These compounds showed transforming activity in a single experiment but not, at any dosage, in five additional experiments. Variable results were also obtained with acetamide, 4-aminobiphenyl, *N*-hydroxy-*N*-2-fluorenylacetamide, and propane sultone; these results could be explained in terms of dosage. The only known carcinogen not inducing transformation was *N*-2-fluorenylacetamide.

- 4838 MALIGNANT LYMPHOMA OR HYDANTOIN LYMPHADENOPATHY? (Ger.) Beil, E. (Dept. Internal Med., U. Munich, Germany) and K. Prechtel. *Munchen Med Wochenschr* 115(45):2033-2039, 1973.

A combination of diphenylhydantoin and phenobarbital was given to a 31-yr-old woman who developed grand mal epilepsy in association with eclampsia. Increasingly frequent seizures were treated with mephenytoin (5-ethyl-3-methyl phenylhydantoin; 0.3 g/day). Pancytopenia, which developed two yr

later after treatment of arthritis with salicylates, aminopyrine and Forapin, was treated with primaclone (total dose 10.5 g) because bone marrow damage was suspected. A sudden decrease in the WBC occurred, but this was reversible when primaclone was discontinued. The patient continued to take mephonytoin for the next eight yr when Hodgkin's sarcoma was diagnosed in the inguinal and axillary lymph nodes. These tumors regressed completely after radiotherapy but recurred in the submandibular glands and axillary lymph nodes about 1-1.5 yr. These recurrences also responded to radiotherapy, but enlargement and thickening of the pulmonary hilus was detected and the left lung eventually collapsed. Severe hemolytic anemia developed 1.5 yr later and the patient died at age 50 yr of cardiovascular failure despite p.o. and parenteral administration of large doses of steroids. At autopsy no evidence was found of systemic malignant lymphoreticular disease. A review of the literature suggests that the phenylhydantoin derivatives which this patient took for epilepsy caused pseudolymphomas, but it is considered likely that these drugs could act as cofactors in the genesis of malignant lymphomas.

- 4839 EFFECT OF PHENOBARBITAL AND DL-ETHIONINE ON 4-(DIMETHYLAMINO)AZOBENZENE-METABOLIZING ENZYMES AND CARCINOGENESIS. (E.) Takamiya, K. (Fac. Pharm. Sci., Chiba U., Japan), S.-H. Chen and H. Kitagawa. *Gann* 64(4):363-372, 1973.

4-(Dimethylamino)azobenzene (DAB) carcinogenesis in rats was delayed by the simultaneous administration of phenobarbital, but accelerated by DL-ethionine. The relationship between the primary metabolisms of DAB in the liver, the cytochrome P-450 content in liver microsomes, and the carcinogenesis in rats receiving DAB alone or DAB plus DL-ethionine or phenobarbital was studied. DAB administration did not influence DAB-metabolizing enzymes or P-450 content throughout the precancerous period. DAB plus phenobarbital promoted activities of the oxidative metabolizing enzyme and P-450 content. DAB plus DL-ethionine markedly depressed the hepatic activity to N-demethylate DAB without a marked influence on other enzymes during the long period of administration. It is suggested that phenobarbital may inhibit DAB carcinogenesis by promoting N-demethylation and hydroxylation. The effect of DL-ethionine on carcinogenesis may result from an increase in proximate carcinogenic metabolites through depression of the N-demethylating activity, or through damage of the N-demethylation system in liver microsomes, or through the action of DL-ethionine as a carcinogenic agent.

- 4840 *SALMONELLA TYPHIMURIUM* *hisG46* (R-Utrecht): POSSIBLE USE IN SCREENING MUTAGENS AND CARCINOGENS. (E.) MacPhee, D. G. (Dept. Genetics and Human Variation, La Trobe U., Bundoora, Australia). *Appl Microbiol* 26(6):1004-1005, 1973.

Two strains of *Salmonella typhimurium* were cultured and placed on agar plates with the mutagen methyl methane sulfonate (MMS): LT2 *hisG46*, which has a

base substitution which alters one codon in the messenger RNA from the gene coding for the first enzyme of histidine biosynthesis; and TA1530, which requires histidine and has a deletion through a gene involved in the excision repair system for DNA. In both LT2 *hisG46* and LT2 *hisG46* carrying the drug-resistance transfer factor R-Utrecht, the addition of between 7.5 and 45 μ mol of MMS caused the appearance of large numbers of revertant (wild type) colonies in a ring around the mutagen. On the addition of 6 μ mol of MMS, two or three times as many revertants of the R-Utrecht strain were found, and on the addition of 3 or 4.5 μ mol, only the R-Utrecht strain yielded revertant colonies. Thus, the R-Utrecht factor appeared to increase the susceptibility of LT *hisG46* to the mutagenic action of MMS and might be expected to increase its susceptibility to the mutagenic effects of compounds which act in a similar way. Trimethyl phosphate also caused the appearance of revertant colonies close to the spot of application in strain LT2 *hisG46* (R-Utrecht) but not in strain LT2 *hisG46* or strain TA1530.

- 4841 DOES RESERPINE INCREASE PROLACTIN AND EXACERBATE CANCER OF PROSTATE? CASE CONTROL STUDY. (E.) Newball, H. H. (Nat'l. Cancer Inst., Bethesda, Md.) and D. P. Byar. *Urology* 11(5):525-529, 1973.

Forty-nine prostatic cancer patients who had been treated with reserpine compounds were compared with 49 similar controls who had not been given reserpine. The subjects were matched in terms of stage of disease, cancer treatment, age, blood pressure, prostatic acid phosphatase, metastasis, activity, pain, and hemoglobin. The no-reserpine group sustained more deaths due to cancer, while the reserpine group sustained more deaths due to cardiovascular disorders: total deaths for the two groups were nearly the same. There were no significant differences in survival, although the reserpine group showed better survival probabilities for 6 yr. A similar pattern was found when the survival experience of 42 of the reserpine patients was compared with that of 809 no-reserpine controls. Thus, there is no evidence that reserpine exacerbates cancer of the prostate either by increasing the serum prolactin or by any other mechanism.

- 4842 SOME BIOLOGICAL EFFECTS OF 7-HYDROXY-METHYL-12-METHYLBENZ(A)ANTHRACENE IN MICE. (E.) Chouroulinkov, I. (Cancer Res. Inst., CNRS, Villejuif, France), A. Gentil and P. Sims. *Biomedicine* 19(10):438-441, 1973.

7,12-Dimethylbenz(a)anthracene (DMBA) and 7-hydroxymethyl-12-methylbenz(a)anthracene (7-OHM-12-MBA) were applied to the backs of female mice, after which the skin was removed for measurement of the epithelial hyperplasia and the disappearance of sebaceous glands. The two compounds were administered over a period of 1 yr to male and female Swiss mice via gastric tube or incorporation into the diet; these mice were then studied for gastric and mesenteric tumors and alterations in the

levels of the hepatic mixed function oxidases. In the skin tests, DMBA showed much stronger carcinogenic activity than 7-OHM-12-MBA. This finding was confirmed in the oral experiments, in which DMBA produced many more stomach and mesentery tumors. However, the two substances produced equal numbers of lung adenomas and other tumors. DMBA in the diet was less active than when administered by tube, and 7-OHM-12-MBA in the diet did not induce any papillomas. The number of lung adenomas was also greater when the compounds were administered by tube. The distribution of the tumors was the same with both compounds. The oral administration of these compounds resulted in an increase in the hepatic microsomal enzyme levels. While the DMBA molecule causes a marked biological effect, it is probable that in the reaction with the intracellular target, metabolic transformation is necessary.

- 4843 INDUCTION OF HEPATOMA IN MICE BY BENZENE HEXACHLORIDE. (E.) Hanada, M. (Osaka U. Sch. Med., Japan), C. Yutani and T. Miyaji. *Gann* 64(5):511-513, 1973.

Six-wk-old dd mice were fed basal diet with 100, 300, or 600 ppm crude benzene hexachloride (BHC) or its α -, β -, or γ -isomers over a period of 32 wk. Exploratory laparotomies performed on the 26th wk revealed liver tumors in 7 out of 26 males and 1 out of 27 females; the β -isomer-treated animals had no tumors. After 38 wk, liver tumors were found in 76.5% of the males and 43.5% of the females, indicating that the males were more susceptible to BHC induced tumors than the females. Again, the β -isomer treated animals had no tumors. The liver tumors consisted for multiple nodules which varied in size and number. No peritoneal invasion or distinct metastasis was found. Microscopically, the tumors appeared to be typical hepatomas with structures similar to that of the normal liver architecture. In addition, foci of enlarged liver cells with nuclear irregularities were scattered in the hepatic lobule. These lesions were also observed in the animals treated with 300 or 600 ppm β -BHC. These proliferative changes of the liver cells were more pronounced in the males than the females. α -Fetoprotein was not detected in the sera of any of the animals with hepatomas. Mammary carcinomas developed in 1 out of 8 members of the 600 ppm α -BHC group and in 2 out of 8 members of the 300 ppm β -BHC group.

- 4844 LIVER TUMOURS AND STEROID HORMONES. (E.) Anonymous. *Lancet* (7844):1481, 1973.

Anabolic and contraceptive steroid hormones can cause various changes in the human and animal liver, including alterations in the bile secretory function, hepatic blood flow, gross morphology, and fine structure of the organ. In addition, there have been several cases in which anabolic and contraceptive steroid hormone treatment has been associated with the development of liver-cell carcinomas, none of them metastatic, and liver-cell adenomas. Although these tumors have all been benign histologi-

cally, they represented a serious threat to the patients; hemoperitoneum, rupture of the liver capsule, and bleeding into the tumor have complicated many of these cases. The development of peliosis in some cases lends support to the idea that the tumors were in some way related to the steroid treatments. While such a relationship is not proven, the critical assessment of any connection between liver tumors and steroid hormones is encouraged.

- 4845 THE IMMUNOSUPPRESSIVE POTENTIAL OF PRODUCTS DERIVED FROM CIGARETTE SMOKE. (E.) Roszman, T. L. (Coll. Med., U. Kentucky, Lexington) and A. S. Rogers. *Am Rev Respir Dis* 108(5):1158-1163, 1973.

The effect of nicotine and water-soluble fraction from whole cigarette smoke on the *in vitro* response to sheep erythrocytes was studied. The results demonstrated that the degree of suppression of the immunoglobulin M and immunoglobulin G antibody responses was dependent on the concentration of either nicotine or water-soluble fraction that was added to the lymphoid cell cultures. A progressive suppression of the immunoglobulin M and immunoglobulin G antibody responses was observed after treatment of these cell cultures with concentrations of nicotine and water-soluble fraction ranging from 1 to 100 μ g/ml. In all cases, the addition to the cultures of either nicotine or water-soluble fraction ranging in concentration from 200 to 1,000 μ g/ml induced complete suppression of both the immunoglobulin M and immunoglobulin G responses. Suppression of these responses was also observed after exposing the cultures to either nicotine or water-soluble fraction for a period of 2 hr before antigenic challenge, indicating that these substances had a rapid and irreversible effect on lymphoid cells. The results presented in this study indicate the immunosuppressive potential of products derived from cigarette smoke.

- 4846 SENSITIVITY OF EMBRYONIC LUNG TISSUE OF A AND C57B1 MICE IN ORGAN CULTURES TO TRANSPLACENTAL ACTION OF URETHANE. (E.) Kolesnichenko, T. S. (Acad. Med. Sci. USSR, Moscow). *Bull Exp Biol Med* 75(5):549-550, 1973.

The transplacental action of urethane in a dose of 80 mg was studied in organ cultures of embryonic lung tissue from mice of lines A (high cancer incidence) and C57B1 (low cancer incidence). Pregnant mice were given s.c. injections of urethane in the last third of pregnancy before the explantation of embryonic lung tissue. Preadenomatous changes were found in the experimental lung tissue culture of A mice: diffuse hyperplasia of the epithelium (36.2%), focal hyperplasia (32.8%), and adenomas (15.5%). Diffuse and focal hyperplasia of the epithelium developed in 39.6 and 8.0% of cases, resp., in embryonic lung tissue cultures of C57B1 mice, but no adenomas appeared. The results indicate correlation between the sensitivity of the embryonic lung tissue of these strains of mice to

the transplacental carcinogenic action of urethane in experiments *in vivo* and *in vitro*. Organ cultures can thus be used as a model to study transplacental carcinogenesis in the lungs.

- 4847 BIOCHEMICAL STUDIES ON CARCINOGENESIS IN THE GLANDULAR STOMACH OF RATS WITH N-METHYL-N'-NITRO-N-NITROSOGUANIDINE. (E.) Saito, T. (Med. Sch., Kyushu U., Fukuoka, Japan) and T. Sugimura. *Gann* 64(4):373-381, 1973.

Studies on DNA synthesis and histochemical studies of leucine aminopeptidase and alkaline phosphatase were performed on the glandular stomach of 60 male Wistar rats treated with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). A solution of 167 µg/ml of MNNG in deionized water was administered freely as drinking water to rats, who were examined at intervals for about 70 wk. One hr before sacrifice, rats were injected i.p. with ³H-thymidine and their tissues were examined by autoradiography and biochemical analysis. Morphologically, three types of stomach lesions were distinguished with different distribution and percentage of labeled cells. The labeling indices of regenerative glandular hyperplasia, upward or downward adenomatous hyperplasia, and adenocarcinoma were 14.0 ± 2.7 , 8.3 ± 1.4 , or 10.4 ± 3.3 and $11.1 \pm 2.6\%$ resp. The rate of DNA synthesis in the whole stomach decreased in the first 20 wk of carcinogenesis, increased from the 20th to 30th wk, corresponding to the repair of erosions or atrophic areas and to the remarkable proliferation observed as regenerative glandular hyperplasia or adenomatous hyperplasia in the body and antral regions, and again decreased after the 30th wk suggesting production of adenocarcinoma in the antral region and atrophy in the body region. Leucine aminopeptidase and alkaline phosphatase activities were observed only in the stroma of the adenocarcinoma. This higher rate of DNA synthesis during regenerative glandular hyperplasia seems important to the production of tumors by MNNG.

- 4848 THE STIMULATORY EFFECTS OF BEARING PRIMARY METHYLCHOLANTHRENE-INDUCED TUMORS UPON THE MURINE LYMPHORETICULAR SYSTEM. (E.) Smith, R. T. (U. Florida Coll. Med., Gainesville) and S. Konda. *Int J Cancer* 12(3):577-588, 1973.

Mechanisms involved in the apparent immunodeficiency associated with the development of primary tumors were studied. Fibrosarcomas were induced in five inbred strains of mice by injecting methylcholanthrene (0.5 mg, i.m.) at four sites. Cellular parameters of spleen-cell immunologic function were measured either after the tumors reached a constant size or following a specific period of growth. T-cell numbers, as indicated by the subpopulation susceptible to anti-θ and complement cytotoxicity, and T-cell functions including PHA mitogenicity, primary alloantigen recognition, and T cooperation were decreased proportionately, although they remained unchanged or were increased absolutely. Susceptibility to LPS mitogenicity, an apparent B-cell function, was increased relatively and

absolutely. The B-cell function of anti-SRBC production was also increased. Colony-forming units increased in number, but were insufficient to account for significant proportions of the spleen cell population. It was concluded that the animal bearing a primary MCA tumor is deficient in neither T nor B cells and that the effect of the tumor upon immune function is stimulatory and similar to the effect evoked by transplanted syngeneic tumors.

- 4849 VARIOUS LEVELS OF DNA REPAIR SYNTHESIS IN XERODERMA PIGMENTOSUM CELLS EXPOSED TO THE CARCINOGENS N-HYDROXY AND N-ACETOXY-2-ACETYLAMINO-FLUORENE. (E.) Stich, H. F. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada), R. H. C. San, J. A. Miller and E. C. Miller. *Nature [New Biol]* 238(79):9-10, 1972.

The levels of DNA repair synthesis in the xeroderma pigmentosum (XP) cells of five patients exposed to the carcinogenic and mutagenic compounds N-acetoxy and N-hydroxy-2-acetylaminofluorene (N-actoxy and N-hydroxy-AAF) was studied. A reduced repair capacity was observed in all cultured XP cells exposed to N-hydroxy-AAF or N-acetoxy-AAF. Some genetic control over the degree of impairment was indicated. The degree of DNA repair synthesis in five XP cell cultures exposed to UV irradiation, 4-nitroquinoline-1-oxide (4NQO), N-hydroxy-AAF, and N-methyl-N-nitrosoguanidine (MNNG) was then studied. The level of DNA repair synthesis was specific for each XP cell culture and was barely affected by the type of initiating agent used. The extent of DNA repair synthesis in the MNNG treated XP cells was comparable to that of the controls.

- 4850 CHEMICAL EVIDENCE FOR THE FORMATION OF A REACTIVE AFLATOXIN B₁ METABOLITE, BY HAMSTER LIVER MICROSOMES. (E.) Garner, R. C. (Dept. Exp. Path. Cancer Res., Yorkshire, England). *FEBS Lett* 36(3):261-264, 1973.

Aflatoxin B₁ was incubated with hamster liver microsomes, after which the microsomes were removed by ultracentrifugation and the fluorescent CHCl₃ extractable metabolites extracted and purified. These metabolites were then compared with the products formed during the reaction of aflatoxin B₁ with m-chloroperbenzoic acid. The latter reaction produced a compound, designated structure II, which, when hydrolyzed with KOH, produced 2,3-dihydrodiolafatoxin B₁; structure II could only have been formed by the initial formation of 2,3-epoxyaflatoxin B₁. Although none of the CHCl₃ extractable metabolites formed during the incubation of aflatoxin B₁ with hamster liver microsomes resembled the dihydrodiol, two water soluble metabolites were formed which appeared to have resulted from further metabolism of the dihydrodiol. Thus, evidence has been provided for the probable formation of 2,3-epoxyaflatoxin B₁ as an intermediate during aflatoxin B₁ metabolism by hamster liver microsomes. It is likely that it is this metabolite which is lethal and mutagenic to bacteria, reacts with nucleic acids, and is the ultimate carcinogenic form of

this compound. Species differences in sensitivity to aflatoxin B₁ carcinogenicity may be related to the balance between metabolism via the epoxide pathway and metabolism through the other known pathway, i.e., activation versus detoxification.

- 4851 INCREASED CARCINOEMBRYONIC ANTIGEN IN HEAVY CIGARETTE SMOKERS. (E.) Stevens, D. P. (Clin. Res. Unit, Walter Eliza Hall Inst. Med. Res., Melbourne, Australia), I. R. MacKay, K. J. Cullen, D. H. Curnow, R. C. Godfrey, M. S. T. Hobbs, M. G. McCall, N. Stenhouse and T. A. Welborn. *Lancet* (7840):1238-1239, 1973.

Sera from 903 persons aged 60 years or more were analyzed by radioimmunoassay for carcinoembryonic antigen (CEA); the smoking habits of these persons were also recorded. All of the subjects were free of clinically detectable cancer. CEA was detected in the serum of 13.6% of the chronic heavy cigarette smokers and in 1.8% of the nonsmokers. A marginally significant increase in the occurrence of CEA among pipe and cigar smokers was noted, and current smokers of fewer than 15 cigarettes per day and former cigarette smokers (regardless of the number smoked per day) did not significantly differ from the nonsmokers in incidence of CEA positivity. The presence of CEA in smokers may be due to precancerous changes, perhaps in the bronchial epithelium. The smoking history is essential in all studies associating CEA with specific diseases and particularly in studies in which CEA is being assessed as a screening test for cancer.

- 4852 TUMORS IN CF-1 MICE EXPOSED FOR SIX CONSECUTIVE GENERATIONS TO DDT. (E.) Turusov, V. S. (Internatl. Agency Res. Cancer, Lyon, France), N. E. Day, L. Tomatis, E. Gati and R. T. Charles. *J Natl Cancer Inst* 51(3):983-997, 1973.

CF-1 minimal inbred mice of six consecutive generations (parents, F₁-F₅) were fed 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (technical DDT) mixed into the diet at dose levels of 2, 10, 50, and 250 ppm for their lifespans. The experiment involved 3987 mice, including DDT-exposed and negative and positive controls. Exposure to all four levels of DDT significantly increased liver tumors (hepatomas) in males (50-55.9% in the 2, 10, and 250 ppm groups and 86% in the 250 ppm DDT groups, compared to 29.5% in controls). In females, hepatoma incidence increased considerably after exposure to 250 ppm DDT (65.5% compared to 4.7% in controls), whereas 10 and 50 ppm DDT only slightly increased the incidence (9 and 13%, respectively). No effect was noted at the level of 2 ppm DDT in females. The average lifespan of males with hepatomas decreased in DDT-treated groups (84 wk at the 250 ppm DDT level, 101-104 wk at the levels of 2, 10, and 50 ppm, as compared to 114 wk in controls). In females, only the highest dose level shortened the average lifespan of hepatoma-bearing mice (94 wk compared to 104 wk in controls). DDT did not alter tumor incidence at sites other than the liver, though an apparent, but not significant, increase in lung tumor incidence was noted at the

levels of 2 and 10 ppm DDT. No progressive increase of hepatoma incidence from generation to generation was noted in DDT-treated mice. However, considerable variations in the incidence of tumors of the liver, lungs, and hematopoietic tissue were observed between the generations within each treatment group, including controls. One metastasizing hepatoma was found in controls and 13 in four DDT-treated groups. Malignant liver tumors, tentatively termed "hepatoblastomas," also occurred, with a slightly increased incidence in the 10 and 50 ppm groups (3.9 and 3.1%, respectively, as compared to 0.9% in controls) and a significant increase in the 250 ppm group (7.1%). Ten of 56 tumors of this type found in DDT-treated mice metastasized to the lungs.

- 4853 ALTERED NUCLEAR RNA TRANSPORT ASSOCIATED WITH CARCINOGEN INTOXICATION IN RATS. (E.) Smuckler, E. A. (Dept. Path., U. Washington, Seattle) and M. Koplitz. *Biochem Biophys Res Commun* 55(2):499-507, 1973.

The release of RNA normally restricted to the nucleus was studied *in vitro* in liver cell nuclei isolated from male Sprague Dawley rats either fed a diet or intubated gastrically with a solution containing the hepatocarcinogen thioacetamide (TIAA) and injected with ¹⁴C-orotic acid prior to sacrifice. ATP-independent loss into the incubation medium of RNA from nuclei isolated 16 or 36 hr after gastric instillation of 5 or 20 mg/100g body weight TIAA was approximately twice as great as that of controls. Loss in the presence of ATP was unaffected. Sucrose density centrifugation of the released RNA indicated that its molecular size was also unchanged. Similar, although accentuated, changes in the rate of loss of nuclear RNA were also seen in tumor-bearing rats fed a diet containing TIAA for 16 wk. Loss of prelabeled DNA from nuclei was unaffected under all experimental conditions. Electron micrographs of the nuclear pellets before and after *in vitro* incubation revealed the presence of an intact nuclear membrane. These results provide further support for the hypothesis that nuclear RNA transport is involved in chemical carcinogenesis.

- 4854 HIGH-FREQUENCY INDUCTION *IN VIVO* OF MOUSE LEUKEMIA IN AKR STRAIN BY 5-AZACYTIDINE AND 5-iodo-2'-DEOXYURIDINE. (E.) Veselý, J. (Inst. Organic Chem. Biochem., Prague, Czech.) and A. Cihák. *Experientia* 29(9):1132-1133, 1973.

The induction of leukemia in the highly susceptible AKR mouse strain by 5-iodo-2-deoxycytidine (IUdR) and 5-azacytidine was studied. IUdR has previously been shown to induce C-type virus formation in cultured Balb/c mouse embryo cells. Female inbred AKR mice received i.p. injections of IUdR (100 mg/kg) or 5-azacytidine (1.5 or 0.8 mg/kg) for a total of 12 inoculations over a 60-day period. Both treated groups became leukopenic after six wk and later developed mild leukocytosis with the appearance of leukemic lymphoblasts. IUdR-treated animals developed leukemia after 50 days of treatment with 67% dying during the next 10 days. All animals receiving 5-azacytidine died of leukemia during the same time

interval. Leukemic cells from both groups were transplantable. Preliminary studies of virus titers in various tissues of 5-azacytidine-treated mice indicated that virus release was being induced under the conditions of the experiment.

- 4855 ESTROGEN-DEPENDENT AND INDEPENDENT RENAL TUMORS. G-6-PD AND LDH ISOENZYME ANALYSIS OF ESTROGEN-DEPENDENT AND INDEPENDENT RENAL TUMORS OF THE SYRIAN HAMSTER. (E.) Dodge, A. H. (Stanford Sch. Med., Calif.). *Oncology* 28(3):253-259, 1973.

Primary renal tumors were induced in golden and albino Syrian hamsters via subpannicular implantation of 30 mg stilbestrol pellets. Autonomous renal tumors derived from the same original primary tumor and spontaneous renal tumors derived from an untreated female were transplanted into golden and Syrian hamster hosts. The primary renal tumors had a glucose-6-phosphate dehydrogenase (G-6-PD) isoenzyme pattern different from that of the autonomous and spontaneous tumors. The form of G-6-PD present in all the tumors had the same RF value as that in red blood cells. The lowest isoenzymes were found in all tumors, with the fastest migrating most prominent in the primary tumors and their metastases but very faint in the autonomous tumors and absent in the spontaneous tumors. When the autonomous tumors were carried in host animals treated with stilbestrol for 30 days, the fastest migrating isoenzymes did not show any appreciable increase. The differences in the lactate dehydrogenase (LDH) patterns were less consistent than those seen with G-6-PD. The spontaneous tumors had an isoenzyme pattern typical of malignant tumors, and the primary tumors and autonomous tumors had a high expression of the fastest migrating isoenzyme with varying expressions of the slowest migrating isoenzyme. LDH and G-6-PD isoenzyme analysis might be useful in clinically distinguishing between hormone-dependent and hormone-independent renal tumors.

- 4856 CADMIUM-INDUCED LEYDIG CELL TUMORS OF RAT TESTIS: MORPHOLOGIC AND CYTOCHEMICAL STUDY. (E.) Reddy, J. (U. Kansas Med. Ctr., Kansas City, Mo.), D. Svoboda, D. Azarnoff and R. Dawar. *J Natl Cancer Inst* 51(3):891-903, 1973.

Nonlethal doses of cadmium salts extensively damage the interstitial tissue of the testis of several mammalian species. Regeneration of the interstitial cells of Leydig ensues in a few months, followed by the unequivocal development of Leydig cells neoplasia. In this study, a single s.c. injection of cadmium chloride (0.03 mmole/kg body wt) induced Leydig cell tumors at one yr in 80% of 30 male Fischer-344 rats. These Leydig cell tumors were studied by electron microscope and histochemical methods. The tumor cells contained abundant agranular endoplasmic reticulum appearing as a network of interconnecting tubules of uniform diameter. The mitochondria were numerous, varied in size, and contained compact vesicular inner structure. In some tumor cells the mitochondrial cristae were not elaborate. The cytoplasmic lipid appeared as numerous vacuoles and was more

prominent in larger tumors. Many microbodies (peroxisomes) were also observed in tumor cells. Some possessed tubular or cylindrical inclusions identical to those in peroxisomes of kidney and spontaneous Leydig cell tumors of the rat testis. Several microbodies also revealed the presence of an amorphous, highly electron dense center. These Leydig cell tumors contained histochemically demonstrable 3 β -hydroxysteroid dehydrogenase activity, which is a key enzyme in steroid biosynthesis.

- 4857 SUBACUTE AND CHRONIC TOXICITY STUDIES OF FLUOROCARBON PROPELLANTS IN MICE, RATS AND DOGS. (E.) Smith, J. K. (Riker Res. Labs., Northridge, Calif.) and M. T. Case. *Toxicol Appl Pharmacol* 26(3):438-443, 1973.

Subacute and chronic inhalation toxicity studies were conducted in rats, mice and dogs treated with fluorocarbon propellants. The fluorinated hydrocarbons tested were trichloromonofluoromethane, dichlorodifluoromethane, dichloromonofluoromethane, monochlorodifluoromethane, trichlorotrifluoroethane, and dichlorotetrafluoroethane. The studies lasted from 2 wk to 23 months and the dose of propellant ranged from 164 to 2240 mg/kg/day. Sedation, ataxia and mild depression were the only signs of toxicity observed, and these occurred in the two studies with the highest propellant doses. These effects, observed during and immediately after exposure, disappeared rapidly. They were attributed to mild anesthetic properties of the fluorocarbon propellants. Hematologic, blood chemistry or urinalysis values were not abnormal in any of the six toxicity studies. Gross and microscopic examination of tissues of the respiratory tract, including lung, did not reveal any evidence of propellant toxicity, irritation or carcinogenicity attributed to inhalation. EKG changes were not observed in dogs receiving large daily doses of the propellants over 3 and 12 months time.

- 4858 SKIN CANCER IN CHRONIC ARSENICISM. (E.) Yeh, S. (Coll. Med., Natl. Taiwan U., Taipei). *Human Pathology* 4(4):469-485, 1973.

The prevalence of skin cancer, hyperpigmentation, keratosis, and blackfoot disease (a peripheral vascular disorder resulting in gangrene of the extremities) among 40,421 residents of a small area of Taiwan where artesian well water with a high arsenic concentration is used was studied. The prevalence rates for these disorders were 1.6%, 18.3%, 7.1%, and 0.89%, resp. The male-to-female ratio was 2.91 for skin cancer, 1.1:1 for hyperpigmentation and keratosis, and 1.99:1 for blackfoot disease; the prevalence of all four increased with age. A total of 303 arsenical cancers in 184 patients were studied histologically; 78% of the patients were 50 yr and older. Of these lesions, 57 were epidermoid carcinoma, 45 were basal cell carcinoma (28 deep and 17 superficial), 176 were intraepidermal carcinoma (23 type B keratosis and 153 classic Bowen lesions and variants), and 25 were combined forms. Bowen's variants included ten with squamous whirls, seven with features of

seborrheic keratosis, and 12 with horn formation. Ten of the basal cell carcinomas revealed bizarre multinucleated giant cells and nuclear atypicalities. Among 81 benign type A arsenical keratoses, 57 revealed no cellular atyp and 24 revealed mild changes. The fine structure in 30 classic cases of Bowen's lesion is described and illustrated. A 5-yr follow-up study of 422 skin cancer patients and 344 patients with blackfoot disease included analysis of mortality ratios and causes of death.

4859 CARCINOGENIC EFFECT OF DI-N-PROPYLNITROSAMINE IN SYRIAN GOLDEN HAMSTERS. (E.)

Pour, P. (U. Nebraska Med. Ctr., Omaha), F. W. Krüger, A. Cardesa, J. Althoff and U. Mohr. *J Natl Cancer Inst* 51(3):1019-1027, 1973.

Weekly s.c. injections of 3.75-60 mg/kg body wt di-n-propylnitrosamine (DPN) caused papillary tumors and carcinomas of the respiratory system in 40 randomly bred Syrian golden hamsters. The trachea was the main target organ, then the nasal cavities, lungs, stem bronchi, and larynx. The number of tumors in the trachea was largest in the upper segment, smaller in the middle segment, and smallest in the lower segment. The low doses of DPN resulted in mostly benign tumors of epidermoid, mucoepidermoid, and polypoid type and a few squamous cell carcinomas in the anterior region of the nasal cavity, whereas the highest doses induced, in addition, carcinomas in the posterior region of the nasoturbinals and ethmoturbinals in 75% of the animals. In general, administration of higher doses of DPN resulted in increased multiplicity of neoplasms despite shorter survival time and tumor latency. In the high dose group the tumors were of different sizes and close together, which probably indicates a more diffuse carcinogenic response of the respiratory epithelium to DPN. Results demonstrate that the carcinogenic response of the Syrian golden hamster to DPN follows distinct patterns in the different segments of the respiratory tract.

4860 METHYLCHOLANTHRENE-INDUCED SARCOMA AND NORMAL MUSCLE FIBROBLASTS OF THE MOUSE--A COMPARISON OF THEIR ULTRASTRUCTURE. (E.)

Heaysman, J. E. M. (Dept. Zoology, U. Coll. London, England) and S. M. Pegrum. *Differentiation* 1(3):191-198, 1973.

The ultrastructure of the normal embryonic mouse muscle fibroblast was compared with that of the methylcholanthrene-induced sarcoma (MCIM) of the mouse; both cell types were cultured *in vitro*. Five main differences between the two fibroblasts were found: the surface of the normal fibroblast was smoother than that of the tumor cell; the leading lamella of the normal fibroblast had fewer ruffles (lamellipodia) and fewer cell-substrate plaques than the tumor cell; the tumor cell did not have a layer of cortical filaments like that of the normal cell, but it did have well marked filamentous tracts in the main body of the cytoplasm; and the endoplasmic reticulum of the normal cell was far less abundant and well-

organized than that of the malignant cell. The fact that all these differences are possibly related to the cell surface may be of significance when considering the changes brought about during carcinogenesis.

4861 EFFECT OF INTRAPERITONEAL PRISTANE ON ESTABLISHED IMMUNITY TO THE Adj-PC-5 PLASMACYTOMA. (E.) Potter, M. (Natl. Inst. Hlth., Bethesda, Md.) and J. L. Walters. *J Natl Cancer Inst* 51(3):875-881, 1973.

A single injection (35 mg/kg/body wt) of *N,N*-di-2-chloroethylaniline [aniline mustard (AM)] into BALB/cAn female mice caused complete regression of large intradermal (i.d.) or s.c. transplants of BALB/c Adj-PC-5 plasmacytoma. After the AM induced regression of the i.d. tumors, 88% of these mice were immune to s.c. challenges of 10^5 viable Adj-PC-5 cells and 73% were immune to i.p. challenges of $\leq 10^4$ viable cells. Also studied was the effect of 1 or 2 i.p. 0.5 ml injections of 2,6,10,14-tetramethylpentadecane (pristane) on the ability of Adj-PC-5-immune mice to resist challenges of Adj-PC-5 cells by various routes. When 10^3 - 10^4 of these cells were injected i.p. into pristane-treated, immunized mice, the tumors grew in 83-100% of the mice, in contrast to only 30% of non-pristane-treated, immunized mice. When 5×10^2 of these cells were injected i.p., the tumors grew in 1 of 10 immune mice and 9 of 13 (68%) pristane-treated, immune mice. Challenges of 20 or 100 cells were eliminated in both pristane-conditioned and normal mice. By contrast, 10^5 Adj-PC-5 cells given s.c. to i.p. pristane-treated, immunized mice were rejected. Apparently pristane directly or indirectly blocked the effectivity of established immunity in the peritoneum but not in the s.c. site and also caused Adj-PC-5 cells to grow more efficiently.

4862 INHIBITION BY CASTRATION OF AFLATOXIN-INDUCED HEPATOMA IN CARBON TETRACHLORIDE-TREATED RATS. (E.) Cardeilhac, P. T. (Dept. Vet. Sci., U. Florida, Gainesville) and K. P. C. Nair. *Toxicol Appl Pharmacol* 26(3):393-397, 1973.

The effect of castration on aflatoxin-induced hepatoma in CCl₄-treated CFN rats was investigated. After 40 days of recovery, rats castrated soon after weaning were given a total of 2.15 mg of aflatoxin B₁-equivalents in 10 doses during a 166-day treatment period. All rats were given 0.1 ml CCl₄ mixed with 0.1 ml of liquid paraffin at the same time treated animals were given aflatoxin. CCl₄ was given to potentiate the effect of aflatoxin. No difference in growth rate was noted between aflatoxin treated rats and controls. Deaths (20-80%) occurred in all groups before the experiment was terminated on day 396. The highest incidence of deaths (80%) occurred in intact rats of both sexes treated with aflatoxin, and hepatomas were found in all four survivors among these animals. No hepatomas were found in any of the 16 aflatoxin-treated surviving castrated rats (39% deaths), in 15 surviving intact control rats (35% deaths), and in 15 surviving cas-

trated control rats (38% deaths). It is suggested that this potentiation may be useful in detecting mycotoxins having a potential for hepatoma induction. These results, along with others, suggest that development and perhaps initiation of aflatoxin-induced hepatoma are potentiated by a functioning gonad.

- 4863 CHANGES IN MICROSOMAL ENZYME ACTIVITIES DURING DAB CARCINOGENESIS. (E.) Meyer, D. I. (Dept. Biol., U. California, Los Angeles) and A. A. Barber. *Chem Biol Interact* 7(4):231-240, 1973.

Microsomal fractions were isolated from the livers of 6-month-old Sprague-Dawley rats which had been maintained for 2 months on normal laboratory chow or a similar diet containing 6% p-dimethylaminoazobenzene (DAB). The membrane-bound activities of glucose-6-phosphatase, NADH cytochrome c reductase, NADPH cytochrome c reductase, and oxidative demethylase were studied, along with the relationship of these activities to membrane composition and conformation. There was a 40 to 60% drop in the activity of all four enzymes in the microsomes of the DAB-fed rats. The DAB feeding also altered the hepatic microsomal membrane in terms of its lipid composition and the interactions between membrane components. The protein components of the membrane were unchanged. There was a 25% increase in the fluorescence of 1-anilino-naphthalene-8-sulfonic acid in the membranes from the DAB-fed animals. Glucose-6-phosphatase was activated by high pH and Triton to a greater extent in the membranes isolated from the DAB-fed rats than in the membranes from the control animals. DAB feeding appears to inhibit the activities of the other three enzymes by a different mechanism than that involved in the inhibition of glucose-6-phosphatase. These enzymes may be affected by direct binding of DAB or its metabolites to enzyme molecules or a DAB effect on the membrane which is not reversible by Triton. The DAB alterations observed herein may explain many of the enzymatic changes observed in the microsomal membranes from carcinogen-fed animals.

- 4864 LIFE-SPAN CARCINOGENICITY TESTS WITH 4-AMINO-N¹⁰-METHYLPTEROYLGLUTAMIC ACID (METHOTREXATE) IN SWISS MICE AND SYRIAN GOLDEN HAMSTERS. (E.) Rustia, M. (U. Nebraska Med. Ctr., Omaha) and P. Shubik. *Toxicol Appl Pharmacol* 26(3):329-338, 1973.

The carcinogenic potential of methotrexate (4-amino-N¹⁰-methylpteroylglutamic acid), a drug used in the treatment of leukemia, psoriasis, and various neoplastic diseases, was tested in a lifetime study in Swiss mice and Syrian golden hamsters. Seven-wk-old mice of both sexes received 10, 8, 5, or 3 ppm of methotrexate in the diet on alternate weeks for life. Seven-wk-old hamsters of both sexes received 20, 10, or 5 ppm of methotrexate in their diet on alternate weeks for life. Decreased survival rates and reduced body wt gain were recorded in the three test groups of hamsters only. The incidence of tumors was not increased in either species. Hepatic

alterations, including hydropic degeneration, fatty metamorphosis and necrosis, were noted for both mice and hamsters. In addition, slight hepatic fibrosis occurred in hamsters and in mice. These histopathologic alterations were spontaneous and consistent with the age and species used. A trend toward an increased rate of malignant lymphomas and lung adenomas was noted in the mice. However, the carcinogenic activity of methotrexate was not conclusively demonstrated. No carcinogenic activity of this antifolate was noted in the hamster tests.

- 4865 INHIBITORY EFFECTS OF LECTINS AND LYMPHOCYTE MITOGENS ON MURINE LYMPHOMAS AND MYELOMAS. (E.) Ralph, P. (Salk Inst. Biological Studies, San Diego, Calif.) and I. Nakoinz. *J Natl Cancer Inst* 51(3):883-890, 1973.

Seven thymus-derived lymphomas in culture were inhibited, as measured by RNA or DNA synthesis, by low concentrations of phytohemagglutinin (PHA), a T-cell mitogen; eight myelomas were resistant to PHA. Both classes of tumors grown in fetal calf serum were inhibited by concanavalin A (Con A), another T-cell mitogen. Higher concentrations of Con A were required to affect cell lines in horse serum, and lymphomas were generally more sensitive than myelomas. Pokeweed mitogen and the B-cell mitogen *Salmonella typhosa* lipopolysaccharide, at concentrations which stimulate lymphocytes to divide, did not affect the tumors. All cell lines were sensitive to ricin, and some IgG2a myelomas were selectively sensitive to extracts of *Ulex europeus*. Inhibition of cellular DNA or RNA synthesis by PHA was blocked by N-acetyl-galactosamine, inhibition by Con A was blocked by methyl-mannoside, and inhibition by ricin was blocked by galactose and partially by N-acetyl-galactosamine. These lectin effects provide alternative markers to cell-surface differentiation antigens for the characterization of T and B lymphocytes and tumors.

- 4866 PATHOLOGY OF INTESTINAL NEOPLASMS AND OTHER LESIONS IN RATS EXPOSED TO AZOXYMETHANE. (E.) Ward, J. M. (Natl. Inst. Hlth., Bethesda, Md.), R. S. Yamamoto and C. A. Brown. *J Natl Cancer Inst* 51(3):1029-1039, 1973.

Male Fischer rats were given 10 weekly s.c. injections of azoxymethane (AOM) at two dose levels, 14.8 and 7.4 mg/kg. Rats were killed at 26 and 34 wk, resp. The number and distribution of intestinal tumors were dose related. Tumors in rats receiving the higher dose were limited mainly to the duodenum and descending colon; few duodenal tumors occurred in the rats given the lower dose. In the latter group, tumors were more generally distributed in the colon. Histologically there were three types of colon tumors: polypoid lesions, adenocarcinomas, and mucinous adenocarcinomas. Carcinomas of both the small and large intestines metastasized to regional lymph nodes and the peritoneal cavity. The pathology of the induced tumors was similar to that of man. Comparison of our results with epidemiologic data and the ratio of ascending

to descending colon tumors in man in high- and low-incidence colon cancer areas revealed a striking similarity to the dose response in this study. Other lesions in rats given AOM included squamous cell carcinoma of the ear canal, hepatic nodular hyperplasia and megalocytosis, and gastric ulcers.

- 4867 PSEUDOCARCINOMATOUS LESIONS OF THE THYROID GLAND AFTER TREATMENT WITH ANTITHYROID DRUGS. (Ger.) Schauer, A. (Pathol. Inst., U. Munich, Germany), E. Kunze and B. Matzner. *Verh Dtsch Ges Pathol* 56:369-373, 1972.
- 4868 A CASE OF A PSEUDOTUMOR OF THE GENITALIA AFTER LONG-TERM TREATMENT WITH ORAL CONTRACEPTIVES. (Pol.) Trzeciak, B. (District Hosp., Kolobrzeg, Poland). *Pol Tyg Lek* 28(5):180-181, 1973.
- 4869 HETEROGENICITY OF THE ANTIGENIC STRUCTURE OF THE LIVER RESULTING FROM ETHIONINE ADMINISTRATION. (Rus.) Kudymov, V. M. (Inst. Nutrition, Moscow, USSR). *Patol Fiziol Eksp Ter* (1):57-59, 1973.
- 4870 THYROID HYPERPLASIA AND PULMONARY TUMORS INDUCED BY METHYLTHIOURACIL AND/OR ISONIAZIDE IN CBA/Cb/Se MICE. (It.) Biancifiori, C. (Inst. Anat. Pathol. Histol., U. Pisa, Italy). *Lav Ist Anat Istol Patol Perugia* 33(1):27-39, 1973.
- 4871 THE CHRONIC TOXICITY OF HEXAMETHYLPHOSPHORAMIDE IN RATS. (E.) Kimbrough, R. D. (Chamblee Toxicol. Lab., Ga.) and T. B. Gaines. *Bull Environ Contam Toxicol* 10(4):225-226, 1973.
- 4872 EFFECT OF N-DIETHYLNITROSAMINE ON EMBRYONIC DEVELOPMENT IN MICE. (Rus.) Petrova-Bergieva, T. (Inst. Industrial Hygiene, Occupational Disease, Sofia, Bulgaria). *Gig Tr Prof Zabol* (10):51-52, 1973.
- 4873 AFLATOXIN B₁ AND PHENOBARBITAL INDUCIBLE AFLATOXIN- Δ^2 -HYDRATION BY RAT LIVER MICROSOMES. (E.) Gilbert, C. (Wellcome Res. Labs., Kent, England). *Biochem Pharmacol* 21(21):2931-2933, 1972.
- 4874 AN INVESTIGATION OF THE GIEMSA BANDING PATTERNS OF MARKER CHROMOSOMES FROM A CLONE DERIVED FROM A RAT TUMOUR. (E.) Scott, S. (Marie Curie Mem. Fdn., Surrey, England), N. P. Bishun and D. C. Williams. *Cytobios* 7(28):207-212, 1973.
- 4875 TISSUE CULTURE OF RAT ASCITES HEPATOMA AH-601 CELLS. (E.) Huh, N. (Inst. Med. Sci., U. Tokyo, Japan), T. Tokaoka and H. Katsuta. *Jap J Exp Med* 42(3):249-262, 1972.
- 4876 METABOLIC ACTIVATION OF MUTAGENS IN MAMMALS. HOST-MEDIATED ASSAY UTILIZING THE INDUCTION OF MITOTIC GENE CONVERSION IN SACCHAROMYCES CEREVISIAE. (E.) Fahrig, R. (Central Lab. Mutagenesis Testing, German Res. Society, Freiburg, Fed. Republic Germany). *Agents Actions* 3(2):99-110, 1973.
- 4877 TRANSPLACENTAL CARCINOGENESIS. (E.) Shapiro, H. A. (Natl. Cancer Assoc., Parktown, Johannesburg, South Africa). *S Afr Cancer Bull* 17(3):81-82, 1973.
- 4878 PRELIMINARY FINDINGS ON THE MUTAGENICITY OF SOME NEW CHEMICAL COMPOUNDS. (Rus.) Movsesian, S. N. (Cytol. Problems Lab., Erevan State U., USSR), M. G. Galukian and R. A. Oganessian. *Biol Zh Armenii* (5):39-43, 1973.
- 4879 RADICAL ACCUMULATION IN LIVER MICROSOMAL MEMBRANES DURING BIOTRANSFORMATION OF AROMATIC AMINES AND NITRO COMPOUNDS. (E.) Stier, A. (Max-Planck-Inst. Biophys. Chem., Gottingen, Germany), I. Reitz and E. Sackmann. *Naunyn Schmiedelbergs Arch Pharmacol* 274:189-191, 1972.
- 4880 THE METABOLISM OF DIBENZO(a,h)PYRENE AND DIBENZO(a,i)PYRENE AND RELATED COMPOUNDS BY LIVER PREPARATIONS AND THEIR ENZYME-INDUCED BINDING TO CELLULAR MACROMOLECULES. (E.) Waterfall, J. F. (Roy. Cancer Hosp., London, England) and P. Sims. *Biochem Pharmacol* 22(19):2469-2483, 1973.
- 4881 THE EFFECT OF CYCLOPHOSPHAMIDE, ³²P AND ⁶⁰Co IRRADIATION ON RABBIT BONE MARROW CELLS. (E.) Kissling, M. (Univ. Leiden Med. Ctr., The Netherlands) and B. Speck. *Blut* 27(3):167-171, 1973.
- 4882 TRANSPLANTABLE MULTIPLE PRIMARY TUMOURS INDUCED IN THE STOMACH, BREAST, OVARY AND ADRENAL OF MICE. (E.) Randeria, J. D. (Cancer Res. Inst., Bombay, India). *Vth Perugia Quadrennial Intl Conference on Cancer* 100, 1973.
- 4883 MULTIPLE PRIMARY TUMOURS IN TRANSPLACENTAL CARCINOGENESIS. (E.) Napalkov, N. P. (USSR Ministry Hlth., Leningrad), V. A. Alexandrov and A. J. Likhachev. *Vth Perugia Quadrennial Intl Conference on Cancer* 99, 1973.
- 4884 MULTIPLE MALIGNANT TUMORS INDUCED IN RATS BY AROMATIC AMINES-RELATED TO N-2 FLUORENYL-ACETAMIDE AND THE DEVELOPMENT OF TRANSPLANTED HEPATOMAS AND KIDNEY TUMORS OF DIFFERENT GROWTH RATE AND BIOLOGICAL CHARACTERISTICS. (E.) Morris, H. P. (Howard U., Coll. Med., Washington, D.C.) and D. R. Meranze. *Vth Perugia Quadrennial Intl Conference on Cancer* 98, 1973.

- 4885 MULTIPLE PRIMARY TUMOURS IN CONVENTIONAL AND GERM-FREE RATS INDUCED WITH THE GLUCOSIDE CYCASIN AND ITS AGLYCONE. (E.) Laqueur, G. L. (Natl. Inst. Hlth., Bethesda, Md.). *Vth Perugia Quadrennial Intl Conference on Cancer* 94, 1973.
- 4886 RELATIONSHIP BETWEEN MULTIPLE PRIMARY TUMORS AND DOSE OF CARCINOGENS, 3-METHOXY-4-AMINOAZOBENZENE AND 1-BUTYL-1-NITROSOUREA, IN THE RAT. (E.) Odashima, S. (Natl. Inst. Hygienic Sci., Tokyo, Japan) and Y. Hashimoto. *Vth Perugia Quadrennial Intl Conference on Cancer* 93, 1973.
- 4887 LEUKEMIA AND MAMMARY CANCER OF RATS ADMINISTERED N-NITROSOBUTYLUREA — ITS SEX AND AGE DEPENDENCY. (E.) Hiroshi, K. (Hokkaido U. Sch. Med., Sapporo, Japan), H. Masuo and G. Eiki. *Vth Perugia Quadrennial Intl Conference on Cancer* 92, 1973.
- 4888 COMPARATIVE MORPHOLOGICAL AND HISTOAUTHORADIOGRAPHIC STUDY OF MULTIPLE EXPERIMENTAL TUMOURS. (E.) Pozharisski, K. M. (N.N. Petrov Res. Ins. Oncology, Leningrad, U.S.S.R.) and V. F. Klimashevski. *Vth Perugia Quadrennial Intl Conference on Cancer* 50, 1973.
- 4889 EXPERIMENTS ON MALIGNANT TRANSFORMATION OF OVARIAN TISSUE IN RATS. (E.) Hilfrich, J. (Med. High Sch., Hannover, Germany) and U. Mohr. *Vth Perugia Quadrennial Intl Conference on Cancer* 48, 1973.
- 4890 VISUALIZATION OF DORMANT NEOPLASTIC POTENTIALITIES IN THE EPIDERMIS DIRECTLY CORRELATABLE TO EVOLUTION OF MULTIPLE SKIN TUMOURS IN MICE. (E.) Telaranta, T. (1st Dept. Path., U. Helsinki, Finland). *Vth Perugia Quadrennial Intl Conference on Cancer* 45, 1973.
- 4891 ELECTRON MICROSCOPIC FEATURES OF THE EPIDERMIS DIRECTLY CORRELATABLE TO EVOLUTION OF MULTIPLE SKIN TUMOURS IN MICE. (E.) Stjernvall, L. (1st Dept. Path., U. Helsinki, Finland). *Vth Perugia Quadrennial Intl Conference on Cancer* 44, 1973.
- 4892 INHIBITION OF EVOLUTION OF MULTIPLE SKIN TUMOURS IN MICE. A SIMPLE TECHNIQUE BASED ON CAUSE AND EFFECT RELATIONSHIPS. (E.) Schreck-Purola, I. (1st Dept. Path., U. Helsinki, Finland). *Vth Perugia Quadrennial Intl Conference on Cancer* 43, 1973.
- 4893 HISTOGENESIS AND CLASSIFICATION OF PULMONARY TUMORS IN MICE. (E.) Kimura, I. (Aichi Cancer Ctr. Res. Inst., Japan). *Vth Perugia Quadrennial Intl Conference on Cancer* 28, 1973.
- 4894 PRENATAL INDUCTION OF BRAIN, KIDNEY, LUNG, LIVER, AND OVARIAN TUMORS IN MICE BY ETHYLNITROSOUREA. (E.) Vesselinovitch, S. D. (U. Chicago, Ill.). *Vth Perugia Quadrennial Intl Conference on Cancer* 91, 1973.
- 4895 MODIFICATION OF ORGANOTROPISM OF N-NITROSOBUTYLUREA-INDUCED CARCINOGENESIS IN MICE BY HOST CONDITIONING. (E.) Yokoro, K. (Res. Inst. Nuclear Med., Biol., Hiroshima U., Japan) and S. Takizawa. *Vth Perugia Quadrennial Intl Conference on Cancer* 90, 1973.
- 4896 TUMOURS INDUCED BY ORAL ADMINISTRATION OF N,N'-2,7-FLUORENYLENEBISACETAMIDE. (E.) Nagayo, T. (Aichi Cancer Ctr., Res. Inst., Japan), M. I. Ito and S. Yamada. *Vth Perugia Quadrennial Intl Conference on Cancer* 89, 1973.
- 4897 COMPARISON OF MULTIPLE SITES OF ATROPHY AND NEOPLASIA INDUCED BY CARCINOGENIC FLUORENYLAMINE COMPOUNDS AND HYDROCARBONS. (E.) Stewart, H. L. (Natl. Cancer Inst., Bethesda, Md.). *Vth Perugia Quadrennial Intl Conference on Cancer* 97, 1973.
- 4898 INFLUENCE OF GRAFT-VERSUS-HOST REACTION (GVHR) ON 7,12-DIMETHYLBENZ(α)ANTHRACENE (DMBA) CARCINOGENESIS IN MICE. (E.) Baroni, C. D. (Inst. Path. Anatomy II, U. Rome, Italy), M. L. Peronace and S. Uccini. *Vth Perugia Quadrennial Intl Conference on Cancer* 96, 1973.
- 4899 MULTIPLE PRIMARY TUMOURS IN RATS TREATED WITH DIMETHYLNITROSAMINE AND ETHYLMETHANESULPHONATE (EMS). (E.) Montesano, R. (Internatl. Agency Res. Cancer, Lyon, France), P. N. Magee and U. Mohr. *Vth Perugia Quadrennial Intl Conference on Cancer* 82, 1973.
- 4900 MORPHOLOGICAL AND BIOLOGICAL BEHAVIOUR OF MULTIPLE PRIMARY MALIGNANT TUMOURS INDUCED IN MICE BY HYDRAZINE SULPHATE. (E.) Cesare, B. (Division Cancer Res., U. Perugia, Italy). *Vth Perugia Quadrennial Intl Conference on Cancer* 81, 1973.
- 4901 COMPARATIVE STUDIES OF THE INFLUENCE OF ALKYL NITROSAMINES ON MAMMALIAN CELLS AND SEA URCHIN EMBRYOS. (E.) Bresch, H. (Med. High Sch., Hannover, Germany) and R. Spielhoff. *Vth Perugia Quadrennial Intl Conference on Cancer* 88, 1973.
- 4902 DISTRIBUTION OF SPONTANEOUS AND CHEMICALLY-INDUCED TUMOURS AT DIFFERENT SITES IN EXPERIMENTS WITH DDT AND URETHANE IN MICE. (E.) Terracini, B. (Natl. Inst. Study, Cure, Tumors, Milan, Italy), R. J. Cabral, M. C. Testa. *Vth Perugia Quadrennial Intl Conference on Cancer* 87, 1973.

- 4903 MULTIPLE INTESTINAL TUMOURS INDUCED IN RATS BY NATURAL CARCINOGENIC PRODUCTS. (E.) Hirono, I. (Gifu U. Sch. Med., Japan) and K. Fushimi. *Vth Perugia Quadrennial Intl Conference on Cancer* 85, 1973.
- 4904 RAPID INDUCTION AND CURE OF LEUKEMIA AND MAMMARY CANCER IN RAT. (E.) Huggins, C. B. (Ben May Lab. Cancer Res., U. Chicago, Ill.). *Vth Perugia Quadrennial Intl Conference on Cancer* 84, 1973.
- 4905 MULTIPLE PRIMARY TUMOURS IN RATS TREATED WITH CERTAIN "NATURAL" CARCINOGENS FROM PLANTS OR FUNGI. (E.) Schoental, R. (Roy. Vet. Coll., London, England). *Vth Perugia Quadrennial Intl Conference on Cancer* 80, 1973.
- 4906 TRANSPLACENTAL INDUCTION OF MULTIPLE PRIMARY TUMORS IN RATS TREATED WITH CHEMICAL CARCINOGENS. (E.) Tanaka, T. (Aichi Cancer Ctr., Res. Inst., Nagoya, Japan) and R. Kinoshita. *Vth Perugia Quadrennial Intl Conference on Cancer* 79, 1973.
- 4907 FACTORS INFLUENCING THE BEHAVIOR OF MULTIPLE INDUCED SKIN TUMORS IN EXPERIMENTAL ANIMALS. (E.) Stenback, F. (Eppley Inst. Res. Cancer, Omaha, Nebr.) and P. Shubik. *Vth Perugia Quadrennial Intl Conference on Cancer* 78, 1973.
- 4908 MULTIPLE CARCINOGENESIS IN THE STOMACH OF RATS, HAMSTERS AND DOGS INDUCED BY N-METHYL-N'-NITRO-N-NITROSOGUANIDINE (MNNG). (E.) Kawachi, T. (Natl. Cancer Ctr. Res. Inst., Tokyo, Japan) and T. Sugimura. *Vth Perugia Quadrennial Intl Conference on Cancer* 77, 1973.
- 4909 OCCURRENCE OF MULTIPLE PRIMARY TUMOURS IN CF-1 MICE. (E.) Breslow, N. E. (Internatl. Agency Res. Cancer, Lyon, France), N. E. Day, L. Tomatis and V. S. Turusov. *Vth Perugia Quadrennial Intl Conference on Cancer* 76, 1973.
- 4910 EUROPEAN HAMSTERS (*CRICETUS CRICETUS* L.) AS A MODEL FOR RESPIRATORY CARCINOGENESIS. (E.) Mohr, U. (Med. High Sch., Hannover, England) and J. Althoff. *Vth Perugia Quadrennial Intl Conference on Cancer* 67, 1973.
- 4911 MULTIPLE PRIMARY TUMOURS IN CYCASIN CARCINOGENESIS. (E.) Fukunishi, R. (Ehime U. Sch. Med., Matsuyama, Japan). *Vth Perugia Quadrennial Intl Conference on Cancer* 95, 1973.
- 4912 A STATISTICAL INVESTIGATION INTO MULTIPLE TUMOURS IN MOUSE SKIN. (E.) Lee, P. N. (Tobacco Res. Council Lab., Harrogate, England). *Vth Perugia Quadrennial Intl Conference on Cancer* 21, 1973.

See also:

- * (Rev): 4802, 4803, 4804, 4807, 4812
- * (Phys): 4918
- * (Immun): 5038, 5116, 5120

- 4913 BIOCHEMICAL EVALUATION OF THE EXTENT OF
EARLY POSTIRRADIATION CELL DAMAGE IN RATS
EXPOSED TO VARIOUS RADIATION DOSES. (Rus.)
Golyshev, Ye. P. (Inst. Biophys., Moscow, USSR) and
T. A. Fedorova. *Biull Eksp Biol Med* (5):44-46, 1973.

Immediately after irradiation with 100, 300 or 900 r from ^{60}Co , adult male albino rats were injected with $2\text{-}^{14}\text{C}$ -orotic acid or $2\text{-}^{14}\text{C}$ -thymidine and urinary excretion of thymidine and deoxyuridine were measured until 8 hr after injection. The specific activity of thymidine excreted in the urine decreased with increasing doses of radiation, while the ratio of ^{14}C -thymidine excreted to that injected increased with increasing doses of radiation. The amount of thymidine synthesized in the body of nonirradiated controls was calculated to be 0.43 mg, while the amount of thymidine synthesized in irradiated rats was 0.66 mg in those given 100 r, 0.89 mg in those receiving 300 r and 1.87 mg in those exposed to 900 r. From previous experiments it was found that the cellular content of DNA was 6.7×10^{-9} mg in adult rats and the number of cells destroyed in 8 hr was about 160 million. In irradiated rats, the number of cells destroyed 8 hr after irradiation was estimated to be 170%, 260% and 630% those of the normal rats.

- 4914 CHROMOSOME ABERRATIONS AS A BIOLOGICAL
INDICATOR OF THE EFFECTS OF RADIATION
AND OTHER ENVIRONMENTAL HAZARDS. (E.) Komarov, E.
(Radiation Hlth., WHO, Geneva, Switzerland). *WHO Chron* 27(11):463-465, 1973.

During the past 10 yr the chromosome aberration test was developed as an indicator to detect the biological effects in persons accidentally or occupationally exposed to radiation. This test is particularly suitable since new techniques now permit chromosome aberrations to be studied in somatic cells using lymphocyte cultures from blood samples. Blood or bone marrow samples must be taken within the first few days or weeks following exposure. The agreement between irradiation levels in different parts of the body and chromosome aberrations in the bone marrow in the same parts of the body is remarkably close. The method may also prove useful for detecting the effects of chemical and other environmental factors on the general population. The significance of the method for detecting the increase in incidence of cancer, leukemia, and other late effects of exposure is not yet fully evaluated. It is suggested that the chromosome aberration rate in somatic cells might be used in studying environmental risks to future generations to evaluate the risk of changes in germinal cells.

- 4915 STIMULATION OF GROWTH OF METASTASES BY
LOCAL X-IRRADIATION IN KIDNEY AND LIVER.
(E.) Van den Brenk, H. A. S. (St. Thomas' Hosp., London, England) and H. Kelly. *Br J Cancer* 28(4):349-353, 1973.

One kidney and the adjacent liver of female, specific pathogen free Caworth Farm rats were lo-

cally X-irradiated with a single 1000 rad dose 7 days prior to the i.v. injection of Y-P388 tumor cells. One day before this injection, some of the rats were given heterologous rabbit anti-rat lymphocytic serum (ALS). Over 200 tumor colonies formed in the lungs of every rat given ALS; in rats given no ALS but injected with five times as many tumor cells, there were fewer colonies. In 15 of the 16 ALS rats and 11 of the 13 no-ALS rats, tumor colonies were more numerous on the surface of the irradiated kidney; the colonies were also larger on the irradiated kidneys. Colony formation appeared to be restricted to the renal cortex. Tumor macrocolonies also appeared in the liver, with the colonies in the irradiated portion of the organ being more numerous and larger. The colonies were more dense than those on the kidneys, but less dense than those in the lungs. The colonies were distributed uniformly throughout the liver. Scattered tumor colonies had also grown under the surfaces of the gastrointestinal tract, mesenteries, thymus and bladder; these colonies were more numerous after ALS treatment.

- 4916 CORRELATION BETWEEN THE RELATIVE QUANTITY
OF SOME NUCLEOTIDE BLOCKS IN DNA OF THE
BODY AND ITS RADIOSENSITIVITY. (Rus.) Kritskii, G.
A. (A. N. Bakh Inst. Biochem., Moscow, USSR) and S.
V. Aleksandrov. *Dokl Akad Nauk SSSR* 212(4):1011-1014, 1973.

- 4917 EFFECT OF THE STAGE IN THE CELL CYCLE AND
TIME FIXATION ON THE INCIDENCE OF CHROMOSOME
ABERRATIONS INDUCED BY RADIATION. (Rus.) Luchnik,
N. V. (Sci. Res. Inst. Med. Radiol., Obninsk, USSR),
K. N. Morozov, E. V. Fesenko and V. A. Lychev. *Dokl Akad Nauk SSSR* 212(5):1220-1223, 1973.

- 4918 LEUKEMIA IN HODGKIN'S DISEASE: IS IT A
RESULT OF CARCINOSTATIC THERAPY? (Ger.)
Schaefer, U. W. (Essen Clin., Germany) and G.
Kanzler. *Med Klin* 67(31):1024-1028, 1972.

- 4919 CARCINOMAS OF THE UPPER URINARY TRACT
AFTER PYELOGRAPHY WITH THOROTRAST. (Fr.)
Ott, R. (Cantonal Hosp., Geneva, Switzerland), R.
Weyeneth and D. Tuchschnid. *J Urol Nephrol* 78(12 bis):225-230, 1972.

- 4920 THOROTRAST: SPECIAL ETIOLOGY OF URINARY
TRACT TUMORS. (Fr.) Petit, R. (Dept.
Urol., U. Liege, Belgium) and J. de Leval. *J Urol Nephrol* 78(12 bis):223-225, 1972.

See also:

- * (Chem): 4881
* (Epid-Biom): 5142

- 4921 INFECTION OF CHICKEN FIBROBLASTS WITH SINGLE EXPOSURE TO DNA FROM VIROGENIC MAMMALIAN CELLS. (E.) Svoboda, J. (Inst. Exp. Biol. Genetics, Czechoslovak Acad. Sci., Prague), I. Hlozanek, O. Mach, A. Michlova, J. Riman and M. Urbankova. *J Gen Virol* 21:47-55, 1973.

The efficiency of the infection of chicken fibroblasts with a single dose (1.5 to 150 µg) of DNA isolated from virogenic Rous sarcoma virus (RSV) (Prague strain) transformed XC cells (a line of rat tumor cells induced with RSV (Prague strain)), was increased if chicken fibroblasts were pre-treated with 5-bromodeoxyuridine (BUDR). Mitomycin (0.1 or 0.01 µg/ml) or u.v. irradiation in doses used (27.6 ergs/mm²/s or 55.2 ergs/mm²s) were not effective. Repeated attempts to transfect duck fibroblasts, not containing activable endogenous chicken virus genome or group-specific antigen, failed. DNA isolated from purified nuclei and from whole virogenic cells exerted the same infecting activity. Infecting activity was present in the peak fractions obtained after CsCl gradient sedimentation of DNA obtained from virogenic cells, was absent in RNA preparations and after digestion of DNA with DNase or alkaline denaturation. This indicates that DNA is responsible for infection.

- 4922 ROLE OF THE CELL FACTOR IN SV40 TRANSFORMATION. (E.) Nachtigal, M. (Acad. Med. Sci., Bucharest, Rumania), N. Sahnazarov and N. Cajal. *Rev Roum Infamicrobiol* 8(3):169-173, 1971.

The characteristics of the following cell types following SV40 induced transformation were studied: Syrian and hybrid (Syrian and Rumanian) hamster kidney, lung, and embryo; and two tumor cell lines obtained by the s.c. inoculation of newborn Syrian hamsters by SV40 virus. All cell lines exhibited unlimited growth capacity *in vitro* following SV40 transformation. In cell lines of tumoral origin, the cellular morphology was fibroblastic. The cell lines of kidney origin took on an epithelial morphology, as did the embryonic cell line and a lung cell line derived from a newborn Syrian hamster. Another lung cell line maintained a fibroblastic morphology, while the third lung cell line maintained a fibroblastic morphology for 12 months then took on an epithelial aspect. Immunofluorescence revealed tumoral antigen (T) within the nuclei of the tumoral, kidney, embryonic, and hybrid hamster lung cell lines; surface antigen (S) was revealed in all but the newborn Syrian hamster lung cell line. There was a complete disappearance of the synthesis of infectious viruses in the transformed cells. The infectious virus was recovered from all but the tumoral cell lines. A particularly high incidence of structural chromosomal aberrations was found in the tumoral cell lines, with a lower proportion of aberrations in the Syrian hamster embryo and kidney cells, and still fewer in the hybrid hamster cells: the number of aberrations in the Syrian hamster lung cell lines did not exceed normal limits. The Syrian hamster

kidney and embryonic cell lines were oncogenic on inoculation to Syrian hamsters. The hybrid hamster cell line induced only one tumor upon inoculation to Syrian hamsters, and the Syrian hamster lung cell lines produced no tumors. Apparently, certain factors synthesized by lung cells suppress certain portions of the SV40 viral genome without being able to prevent cellular transformation by this virus.

- 4923 *IN VITRO* TRANSFORMATION OF HAMSTER EMBRYO CELLS BY A GUINEA PIG HERPES-LIKE VIRUS. (E.) Fong, C. K. Y. (Yale U. Sch. Med., New Haven, Conn.) and G. D. Hsiung. *Proc Soc Exp Biol Med* 144(3):974-978, 1973.

Hamster embryo cell cultures were infected with guinea pig herpes-like virus (GPHLV) strains LK31, LK40, LK51, H-62, H-114, and H-125, or with UV-inactivated virus preparations. After 3 wk, 10 to 50% of the infected cultures developed morphologically transformed foci. Cell lines cultured from these foci contained primarily epithelial-like cells, with some fibroblast-like cells and cells with enlarged nuclei. Both infectious and UV-inactivated virus preparations isolated from leukemic guinea pigs induced noticeable cell transformation in 72% of infected cultures; only 45% of the cultured infected with virus preparations from nonleukemic animals showed cell transformation. Viral specific antigen was found in 7 to 20% of the transformed cells, but neither herpesvirus nor C-type virus particles could be detected in these cells. GPHLV was recovered from a very small number of cells in 7 of 8 cell lines tested. The ability of GPHLV to transform hamster cells is probably due to the herpesvirus infection.

- 4924 ONCOGENIC MAREK'S DISEASE HERPESVIRUS IN AVIAN ENCEPHALITIS (TEMPORARY PARALYSIS). (E.) Kenzy, S. G. (Agricultural Res. Ctr., Washington State U., Pullman), B. R. Cho and Y. Kim. *J Natl Cancer Inst* 51(3):977-982, 1973.

When oncogenic Marek's disease herpesvirus (MDHV) was present in blood or tissue culture inocula, an encephalitis designated as temporary paralysis (TEP) could be consistently reproduced in White Leghorn chicks. It resembled transient paralysis (Zander) and the temporary paralysis syndrome (Walker and Grattan). Mild MDHV and turkey herpesvirus (HVT) failed to produce a TEP response. This response was effectively reduced by pretreatment of chicks with HVT or mild MDHV 8 days before inoculation with blood from acute MD-affected fowl, by rapid freezing and thawing of infective blood before inoculation into chicks, and by inoculation with sera from MD survivors 4 or 5 days before inoculation with oncogenic MDHV. TEP was also observed in chicks exposed to airborne dust from chickens with MD, although the level of TEP response was low compared to inoculated chicks. Chicks with TEP developed a concurrent viremia, due to MDHV, and microscopic neural lesions of MD. Some chicks died while paralyzed, but most chicks recovered rapidly, only to die 2-6 wk later

from clinical MD. No evidence of any encephalitic bacterial or viral agent other than MDHV was obtained.

- 4925 SPONTANEOUS *HERPESVIRUS SAIMIRI* LYMPHOMA IN AN OWL MONKEY. (E.) Hunt, R. D. (Harvard Med. Sch., Southborough, Mass.), F. G. Garcia, H. H. Barahona, N. W. King, C. E. O. Fraser and L. V. Melendez. *J Infect Dis* 127(6):723-725, 1973.

Two cases of spontaneous malignant lymphoma in owl monkeys occurred within a 3-month period. *Herpesvirus saimiri* was isolated from peripheral lymphocytes, kidney, and liver of one animal. Microscopic findings included diffuse sheets of lymphocytes and lymphoblasts in liver, spleen, lymph nodes, kidney, myocardium, pancreas, cervical muscles, and bone marrow. These findings provide, for the first time, evidence that *H. saimiri* can cause spontaneous lymphoma in owl monkeys and that a herpesvirus-induced malignancy of mammals can be a contagious disease.

- 4926 CHARACTERIZATION OF C-TYPE PARTICLES PRODUCED BY A TISSUE CULTURE-ADAPTED MURINE MYELOMA. (E.) Volkman, L. E. (Dept. Microbiol., U. Washington, Seattle) and R. G. Krueger. *J Virol* 12(6):1589-1597, 1973.

C-type particles produced by a tissue culture-adapted BALB/c myeloma were characterized. Although the particles were morphologically and antigenically similar to murine leukemia (MuLV) and sarcoma virus, the size of their RNA was different, they lacked RNA-dependent DNA polymerase, they were unstable in NET buffer, sucrose and citrate but were stable in glycerol and Earle balanced salt solution, and they behaved differently from oncornaviruses when treated with ether and detergent. These particles therefore appear to represent either an MuLV subtype, a defective MuLV virion, or a new murine virus.

- 4927 HORIZONTAL TRANSMISSION OF LEUKEMIA VIRUS AND LEUKEMIA IN THE CAT. (E.) Jarrett, W. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.), O. Jarrett, L. Mackey, H. Laird, W. Hardy, Jr. and M. Essex. *J Natl Cancer Inst* 51(3):833-841, 1973.

Neonatal and adult cats were exposed to cats that had been inoculated with feline leukemia virus (FeLV). A range of techniques was used on both sets of animals to monitor the presence or absence of virus, group-specific antigen, and antibodies to the virus and feline oncornavirus-associated cell membrane antigen (FOCMA). Horizontal transmission, occurring in most cases, took place between littermates when only a fraction were inoculated at birth and also when litters inoculated neonatally were housed from birth with unrelated litters. Adults became infected when exposed to cats that had been inoculated with FeLV as adults. Transfer of infection was demonstrable within a month of mixing;

transmission of virus in this way could lead to the development of tumors in cats exposed as kittens or as adults. A wide variety of tissues was examined electron microscopically to determine possible routes of excretion of FeLV. Virus was most common in the trachea and oral mucosa, but it was also recovered from the urine and urinary bladder. Four adult cats became infected after being housed for 1 week with a cat with a naturally occurring alimentary lymphosarcoma. These results do not exclude the possibility of vertical transmission of virus, but they do indicate that horizontal transmission is an important epidemiologic factor. Thus, caution should be applied in taking the mouse, where vertical transmission is interpreted as the only important route, as a model for human leukemias.

- 4928 RETINOBLASTOMA-LIKE TUMORS INDUCED IN RATS BY HUMAN ADENOVIRUS. (E.) Kobayashi, S. (Retina Fdn., Boston, Mass.) and N. Mukai. *Invest Ophthalmol* 12(11):853-856, 1973.

Newborn rats (35) were given a single injection of 0.01 ml of the human adenovirus Type 12 fluid into the vitreous. The supernatant of non-virus-infected HeLa cells was injected into the vitreous of control animals. The first animal produced a massive intraocular tumor 204 days after injection. By day 300, 3 of the 35 rats produced similar intraocular malignant tumors. None appeared in control rats. Extensive studies of these tumors showed them to be retinoblastoma-like. It is assumed that the malignant tumors produced by adenovirus Type 12 are derived from neuronal precursor cells of the developing retina, and are therefore considered as a counterpart of retinoblastomas without rosettes in the human.

- 4929 ISOLATION OF A RIBONUCLEOPROTEIN STRUCTURE FROM ONCORNAVIRUSES. (E.) Fleissner, E. (Sloan-Kettering Inst., New York, N.Y.) and E. Tress. *J Virol* 12(6):1612-1615, 1973.

Treatment with Nonidet P-40 was used to isolate ribonucleoprotein (RNP) from MC29 avian leukosis virus (AvLV), avian myeloblastosis virus (AMV), and the Rauscher strain of murine leukemia virus (MuLV). Gel filtration of an MC29 AvLV RNP preparation revealed the presence of one major protein species corresponding to the p12(N) viral component and representing 10% of the virion protein. Minor species in the 60,000 to 90,000 molecular weight range were also observed when processing large quantities of virus. Gel filtration of an MuLV preparation also revealed only one major protein species, correspondent to the smallest viral structural protein (p10(n)). For both the MC29 AvLV and MuLV viruses, the RNP-associated protein was the most arginine-rich of the virion internal proteins. For AMV and MC29, 30 to 50% of the reverse transcriptase originally present in the virions could be recovered in RNP preparations. In RNP preparations from MuLV, about 10% of the original enzyme activity was recovered, the remainder of the activity being found in the detergent layer of the sucrose gra-

dient. Thus, the association of the enzyme with the RNP structure appears to be less stable to detergent for MuLV than it is for AvLV. The oncornavirus RNPs contained a much higher percentage of RNA (20 to 25%) than do the RNPs of influenza, parainfluenza, and rhabdoviruses.

- 4930 INDUCTION OF SIMIAN VIRUS 40 (SV40) IN TRANSFORMED HAMSTER CELLS WHICH ARE SENSITIVE AND RESISTANT TO ACTINOMYCIN D. (Fr.) Suarez, H.-G. (Inst. Sci. Res. Cancer, Villejuif, France) and R. Cassingena. *C R Acad Sci [D]* (Paris) 277:1269-1272, 1973.

Trypsinized cells from *Cercopithecus* monkeys (CV-1) were mixed with equal parts of hamster cells transformed by SV40 and transformed hamster cells resistant to 1 µg/ml actinomycin D in the presence or absence of activated Sendai virus. In both the presence and absence of Sendai virus actinomycin D-sensitive cells fused with CV-1 cells 3-4 times more frequently than did actinomycin D-resistant cells. This may be due to changes which occurred in the cytoplasmic membrane of the resistant cells when they acquired resistance to actinomycin D. However, the proportion of V-SV40-positive heterokaryons appeared to be about the same in cultures of both sensitive and resistant cells. Sensitive cells contained 130-3300 times more infectious viral DNA and 60-550 times more virions than did resistant cells incubated with CV-1. These differences cannot be accounted for by the greater tendency of nonresistant cells to fuse. SV40 induced in resistant cells formed small-plaque mutants which were less capable of replicating in CV-1 cells than SV40 (large plaque) induced in nonresistant cells.

- 4931 A SCANNING ELECTRON MICROSCOPE STUDY OF SURFACE FEATURES OF VIRAL AND SPONTANEOUS TRANSFORMANTS OF MOUSE BALB/3T3 CELLS. (E.) Porter, K. R. (Dept. Molecular, Cellular, Developmental Biol., U. Colorado, Boulder), G. J. Todaro and V. Fonte. *J Cell Biol* 59(3):633-642, 1973.

The surface features of cells from the following lines were studied via electron microscopy: BALB/3T3, clone A31; an SV40-transformed subclone derived from it (SV-T2); a murine sarcoma virus transformant of A31 (K-A31); a mouse polyoma virus transformant (Py-3T3-4a); an *in vitro*-selected spontaneously transformed variant (S2-4); and an *in vivo*-selected spontaneous transformant (TuT3). The parent cell in confluent culture closely resembled an endothelial cell in its form and in the structure of its association with adjacent cells. The tumorigenic virus-induced transformants were fusiform to pleomorphic and distinctly different from the parent cell. They presented relatively smooth surfaces except for blebs and marginal microvilli and closely resembled each other. However, the virus-induced transformants differed in form from the tumorigenic transformants of spontaneous origin. The S2-4 cells, which showed the most extreme departures from the parent cells, possessed a thickened rather than the lamellar form of the parent A31 cells and were covered by long microvilli and many spherical blebs. The TuT3 cells more closely resembled the parent cells but showed extensive ruffling at their margins. All transformants grew without evidence of contact inhibition.

It is concluded that the progeny of a single cell can be altered by both extrinsic and intrinsic factors.

- 4932 ONCOGENICITY OF LYMPHOID CELLS IMMUNE TO MOLONEY'S MURINE SARCOMA. (It.) Giuliani, F. (Natl. Inst. Tumor Res. Treatment, Milan, Italy), C. Soranzo, A. M. Casazza and A. Di Marco. *Tumori* 59(4):269-276, 1973.

Moloney's murine sarcoma virus (MSV-M) was inoculated i.m. into 30-day-old BALB/c mice. After 24-27 days, when the tumors had regressed, a suspension of cells from the spleen and lymph nodes of these animals were inoculated i.p. into syngeneic mice. Tumors developed in 25 of the 27 inoculated mice. In contrast, tumors developed in only 2 of 8 inoculated mice which had previously been injected with MSV-M. Tumors in immune mice were similar to the rhabdomyosarcomas induced by i.m. inoculation of MSV-M. The tumorigenicity of extracts of lymphoid cells from immunized mice varied. In some cases a virus was isolated which induced foci of transformation in cultures of embryonic fibroblasts from Swiss NIH mice *in vitro* and induced tumors *in vivo*. In one case, however, extracts from lymphoid cells of immunized mice failed to transform embryonic mouse fibroblasts, even after many passages and in the presence of Moloney's murine leukemia virus; they failed to induce tumors when inoculated i.m. into adult and newborn BALB/c mice. Virus was isolated *in vitro* from tumor extracts.

- 4933 COMPARISON OF THE YIELD OF INFECTIOUS VIRUS FROM CLONES OF HUMAN AND SIMIAN LYMPHOBLASTOID LINES TRANSFORMED BY EPSTEIN-BARR VIRUS. (E.) Miller, G. (Yale U. Sch. Med., New Haven, Conn.) and M. Lipman. *J Exp Med* 138(6):1398-1412, 1973.

Three lymphoblastoid cell lines of human (883L), squirrel monkey (B84-15), and marmoset (B95-8) origin, all of which had been transformed by the same strain of Epstein-Barr virus (EBV), differed markedly in their content of infectious virus. Single cell clones were obtained from each line to determine whether these differences were dependent upon factors shared by all cells in each line or upon factors present only in a proportion of the total cell population. A total of 17 primary clones representing all three species were examined. Cloning efficiency on human placental cell feeder layers varied from 16 to 24%. EBV antiserum, present in the cloning suspension, neutralized all extracellular viruses. Fifteen of the 17 clones released EBV as measured by a transformation assay. Titers of infectious virus released by daughter clones paralleled titers of virus in the parent line. The marmoset clones yielded the highest titers of infectious virus, while the human clones yielded the lowest. The median yield of virus from the clones of all three species was 4 (human), 96 (squirrel monkey), and 786 (marmoset) transforming units per 1000 cells containing viral capsid antigen. Two nonproducer clones (human and squirrel monkey) did not release infectious virus after

treatment with 5'-bromodeoxyuridine or with X-ray followed by co-cultivation with marmoset leukocytes. The nonproducer clones could not be superinfected by biologically active EBV. Thus, differences in the production of infectious EBV among the lines tested are reflected in the majority of cells of these lines. It appears that the mechanism for regulation of the expression of the EBV genome is cellular rather than viral in origin. There are presumably genetic differences among primate species in this regulatory process.

- 4934 SIZE DIFFERENCES IN THE RIBONUCLEIC ACIDS OF FELINE LEUKAEMIA VIRUSES. (E.) Whalley, J. M. (Leukaemia Res. Unit, U. Glasgow, Scotland). *J Gen Virol* 21(1):39-46, 1973.

Acrylamide and agarose gels were used to compare the sizes of RNAs from the following feline leukemia virus (FeLV) isolates: FeLV-1, FeLV-5, and FeLV-A, B, and C. Electrophoretic analysis of the native and heat-denatured RNAs from these isolates revealed small but significant size differences. The molecular wt of the denatured RNAs from the FeLV subgroups A, B, and C were estimated to range from 2,200,000 (FeLV-A) to 2,600,000 (FeLV-B). The RNA of the A virus was smaller than that of the B virus, while that of the C virus appeared to be intermediate in size between the other two. Only a slight difference was observed between the subunit RNAs of FeLV-1 (A subgroup) and FeLV-5 (a mixture of A and B subgroups), the latter appearing to move more slowly during gel electrophoresis. However, since the RNA of FeLV-A migrated distinctly more rapidly than that of FeLV-1, although both viruses are of the same subgroup, no simple relationship exists between RNA size and virus subgroup. The size difference between the subunit RNAs of the various FeLV isolates could mean the difference in ability to code for 1 to 3 proteins, possible envelope glycoproteins.

- 4935 THE RELATIONSHIP OF POLYOMA VIRUS-INDUCED TUMOR (T) ANTIGEN TO ACTIVATION OF DNA SYNTHESIS IN RAT MYOTUBES. (E.) Graessmann, A. (Inst. Physiol. Chem., Free U. Berlin, West Germany), M. Graessmann and M. Fogel. *Dev Biol* 35(1):180-186, 1973.

Rat myotubes (multinucleated muscle fibers) infected with polyoma virus (PV) introduced into the multinucleated cells by virus-bearing myoblasts at the time of cell fusion incorporate ^3H -TdR and exhibit mitotic-type figures. The infected myotubes also produce a viral-specific nuclear antigen, tumor (T) antigen, which appears in groups of adjacent nuclei or in all nuclei of the myotubes. The proportion of myotubes which synthesize DNA, T-antigen and exhibit mitotic-type figures is related to the multiplicity of virus infection. Intact myotubes which are resistant to infection with PV by virus absorption can be infected by microinjection of the virus into the cells. Myotubes thus infected produce T-antigen which appears in multiple nuclei, but do not incorporate ^3H -TdR or contain mitotic-type figures. The data suggest that the re-

sistance of myotubes to infection with PV might be due to a change in the cell surface membrane during differentiation so that virus cannot penetrate the cell. The T-antigen apparently has no bearing on the activation of the DNA-synthesizing apparatus in multinucleated muscle cells.

- 4936 PATHOGENESIS OF FELINE VIRAL FIBROSARCOMAS: DOSE AND AGE EFFECTS. (E.) Snyder, S. P. (Dept. Vet. Med., Oregon State U., Corvallis) and D. L. Dungworth. *J Natl Cancer Inst* 51(3):793-798, 1973.

The effects of virus dose and host age at the time of inoculation were studied in cats. Twenty-eight neonatal kittens were inoculated with tenfold serial dilutions of a pooled cell-free preparation of ST-feline sarcoma virus to examine the effects of virus dose on subsequent tumorigenesis. Similarly, age effects were studied in 20 cats ranging from neonates to 1-year-olds. Fibrosarcomas induced in younger animals and in those given the highest doses of inoculum (10^0) had shorter latent periods, enlarged rapidly, contained more virus particles on electron microscopy, attained greater size, and resulted in the animals' deaths. Conversely, older animals and those given lower doses of inoculum (10^{-1} , 10^{-2} , 10^{-3}) developed slowly enlarging tumors after a prolonged latent period; these fibrosarcomas contained fewer virus particles and tended to regress. Occurrence of metastases was related to initiating virus dose. At the 10^0 dose level, the animals died before metastases became obvious grossly. Metastases were more common in 10^{-1} and 10^{-2} dose groups; they were uncommon in the 10^{-3} group. In kittens developing metastases, there was no dose-related site distribution of lesions.

- 4937 DIFFERENCES IN MURINE LEUKAEMIA VIRUS-SPECIFIC DNA SEQUENCES IN NORMAL AND MALIGNANT CELLS. (E.) Viola, M. V. (Howard U. Sch. Med., Washington, D. C.) and L. R. White. *Nature* 246(5434/5):485-487, 1973.

Radioactively labeled single stranded DNA copies of the naturally occurring AKR murine leukemia virus (AKR-V) and the Rauscher leukemia virus (RLV) were synthesized *in vitro* by an endogenous reverse transcriptase reaction. Cell DNA extracted from normal cells of low (BALB/c, NIH Swiss) and high (AKR) leukemia incidence mouse strains and Rauscher and Gross Virus-induced lymphomas were then hybridized with these DNA copies. AKR- ^3H -DNA hybridized more extensively with all mouse DNA preparations than did RLV- ^3H -DNA. The percentage of AKR probe hybridized was greatest with lymphoma DNA and successively less with normal AKR, BALB/c, and NIH Swiss DNA. RLV- ^3H -DNA hybridizations showed no differences between mouse strains, although it hybridized more extensively with lymphoma DNA than with normal DNA. The AKR probe reannealed more rapidly with lymphoma and normal AKR DNA than with NIH Swiss DNA. The duplexes formed by AKR- ^3H -DNA with lymphoma and AKR DNA showed higher thermal stability than those formed with BALB/c and NIH Swiss DNA. The differences in the

extent of hybridization and reannealing kinetics among the DNA preparations tested indicate quantitative differences in the murine leukemia virus information in the cell DNA; the differences in thermal stability indicate qualitative differences.

- 4938 SPECIFIC KILLING OF RSV-TRANSFORMED CELLS *IN VITRO* BY LYMPHOID CELLS FROM RSV TUMOR-BEARING CHICKENS. (E.) Ben Sasson, Z. (Lautenberg Ctr. Gen. Tumor Immunol., Jerusalem, Israel), F. Doljanski and D. W. Weiss. *Isr J Med Sci* 9(3):258-265, 1973.

Lymphoid cells derived from the spleen, thymus, or blood of chickens carrying sarcomas induced by the Rous sarcoma virus (RSV) exhibited specific cytotoxic activity against chicken embryo cells transformed by RSV in 9 out of 21 experiments. Cytotoxic capacity was measured by the release of ^{51}Cr from the target cells *in vitro*. In no instance did lymphocytes from tumor-bearing chickens display a significantly greater cytotoxic reactivity against normal than against transformed chicken embryo cells and in no instance were lymphoid cells from normal chickens more reactive against the transformed than against the normal target cells. The specifically heightened cytotoxic capability of lymphocytes from birds exposed to the tumors is interpreted as indicating a state of heightened host resistance to the neoplasm. Possible reasons for the only limited incidence of the *in vitro* cellular cytotoxic (immune) reactivity are interference by other cell surface constituents, the transience of cellular immune elements during active growth of Rous sarcomas, and the lower *in vitro* sensitivity of the chromium release assay as compared with the colony inhibition technique. Another possibility is inhibition of lymphoid cell-target cell interactions by shed plasma membrane constituents of neoplastic cells.

- 4939 CYTOLOGIC ASPECTS OF ABORTIVE TRANSFORMATION IN THE POLYOMA VIRUS-HAMSTER CELL SYSTEM. (E.) Robert-Vague, D. (Nat'l. Inst. Hlth. Med. Res., Marseille, France), H. P. Bonneau, G. Ingenito and H. Bonneau. *Acta Cytol (Baltimore)* 17(6):487-492, 1973.

Cultures of baby hamster kidney cells of the BHK 21/C13 line were infected with polyoma virus of the Small Plaque Toronto strain. At the 6th hr post-infection, a basophilic cytoplasm, some cytoplasmic overlapping, and moderate nuclear increase with respect to the cytoplasm was evident. After 12 hr, a less homogeneous culture with some free cells and a diminished contact inhibition was noted. After 18 hr, there was an extensive overlapping of the cells at both the cytoplasmic and nuclear levels with enormous nuclear abnormalities and cytoplasmic inclusions and numerous free degenerated cells. After 21 hr, the culture had degenerated even more, and by the 8th day, a very poor culture with two cell populations was obtained. No significant cytologic difference was found between cells cultured in serum rich and serum poor media. The cultures infected with a multiplicity of 200 plaque-forming units (PFU) showed

more marked degenerative aspects than the cultures infected with 100 PFU/cell.

- 4940 CHARACTERISTICS OF *HERPESVIRUS SAIMIRI*-INDUCED LYMPHOMA CELLS IN TISSUE CULTURE. (E.) Rabin, H. (Litton Bionetics, Inc., Kensington, Md.), G. Pearson, H. C. Chopra, T. Orr, D. V. Ab-lashi and G. R. Armstrong. *In Vitro* 9(2):65-72, 1973.

Lymphoblastoid cells were cultured from two *Herpesvirus saimiri* (HVS) inoculated white-lipped marmosets and from one HVS-inoculated owl monkey. Cells from all three animals grew clumped in suspension and did not settle on the surface of the culture vessel. Cells from both species were diploid in chromosome number and had no unusual chromosomal abnormalities. The marmoset cell line was chimaeric. The marmoset cells lacked HVS-associated antigens as determined by immunofluorescence. No evidence was found for the presence of virus in the marmoset cells by either assays or electron microscopy. Cocultivation of these cells with Vero cells caused cytopathology and the recovery of complete, infectious virus. On the other hand, the owl monkey lymphoid cells were positive to a small degree both for viral antigens and infectivity. The cells resisted rechallenge with HVS. Cocultivation of these cells with Vero cells caused cytopathology and increased the yield of virus. Results of these studies show that similarities exist between the HVS-carrying lymphoblastoid cells and human lymphoblastoid cells carrying EBV, which in turn supports the theory that HVS is a valid model for studying possible herpes type virus-induced neoplasms of man.

- 4941 PASSAGE OF A VIRAL LEUKEMIA-LIKE DISEASE AND LEUKOSIS TO MONKEYS (*MACACA SPECIOSA*) FROM MONKEYS INOCULATED WITH BLOOD FROM PATIENTS WITH LEUKEMIA. (Rus.) Lapin, B. A. (No affiliation), L. A. Iakovleva, L. V. Indzhia, M. I. Kuksova, L. V. Kokosha, I. A. Bukaeva, M. T. Ivanov, V. F. Shekolodkin, V. N. Lebedev, A. F. Bykovskii and Iu. I. Lor'e. *Vest Akad Med Nauk SSSR* (4): 10-20, 1973.

Monkey passages have been made of whole blood and plasma filtrates from two monkeys (*Macaca speciosa*) which had been inoculated with blood from two patients with acute leukemia. One of these monkeys died one month after inoculation with no clinical symptoms, while the other died after 2 yr 4 months with clinical symptoms of a mild, recurrent leukemia-like disease. Leukemia developed in recurrent and abortive forms in monkeys injected i.v. with passaged whole blood and plasma from these two monkeys. The indirect immunofluorescence test revealed that WBC from both monkeys inoculated with blood from leukemia patients and monkeys receiving whole blood and plasma passages contained a surface antigen which was not present in healthy monkeys or monkeys which had been inoculated with blood from normal humans or monkeys. This surface antigen persisted for 2-3 yr in monkeys with severe leukemia and disappeared within 2-3 months after inoculation

in those with abortive forms of the disease. Type C virus particles were detected in four 48-hr cultures of WBC; these particles were similar to those found in one of the monkeys inoculated with blood from a leukemia patient. It is considered that the leukemia-like disease was induced in monkeys by human leukemia virus, rather than by monkey leukemia virus.

- 4942 FRIEND LEUKEMIC MOUSE STEM CELL REVERSION TO NORMAL GROWTH IN IRRADIATED HOSTS. (E.) Mاتيولي, G. (U. Southern California Sch. Med., Los Angeles). *J Reticuloendothel Soc* 14(4):380-386, 1973.

Injection of Friend leukemic virus (FLV) into adult DBA/2j mice caused rapidly progressive splenomegalic erythroleukemia. Friend leukemic stem cells (FLC) increased at a doubling time of about 34 hr for 7 or 8 days, and then slowed until day 25 to 30 when the leukemic mice died. The self-renewal and differentiation probabilities of the FLC growing in leukemic spleen were greatly abnormal, perhaps due to inefficient differentiation of incompletely maturing erythroid cells. A preparation of FLC from Friend leukemic spleens was tested in lethally irradiated hosts by the recovery method and an almost complete reversion of the FLC to normal renewal and differentiation was observed. It was noted that FLC and normal stem cells are equally efficient in restoring the hematopoietic functions of the irradiated host. The FLC renew and differentiate stochastically as the normal stem cells. Experimentation ruled out the possibility of both normal and leukemic stem cells existing side by side in the leukemic mice, and showed that every single colony was virus infected and formed by an original "leukemic" stem cell. It is concluded that the vast majority of the stem cells growing in irradiated mice are the original virus-infected "leukemic" stem cells from the leukemic donor. Therefore, the "leukemic" condition for certain stem cells is fundamentally metastable and dependent on the hematopoietic microenvironment to which stem cells must adapt in order to grow.

- 4943 EVIDENCE FOR 30-40S RNA AS PRECURSOR OF THE 60-70S RNA OF ROUS SARCOMA VIRUS. (E.) Duesberg, P. (Dept. Molecular Biol., U. California, Berkeley), E. Canaani and K. von der Helm. *Am J Clin Pathol* 60(1):57-64, 1973.

Rous sarcoma virus (RSV) harvested from infected cells at intervals of 3 minutes has the same density, sedimentation coefficient, and DNA polymerase as virus harvested at hourly intervals. The RNA of such RSV consists of a minor class of 60-70S RNA, a major class 30-40S RNA, and a 4-12S class of RNA present in variable concentrations. Upon incubation of the virus harvested at 3-minute intervals at 40°C in cell growth medium or Tris-saline, most of the 30-40S RNA is converted to 60-70S RNA. The electrophoretic mobility of the 30-40S RNA of RSV harvested at 3-minute intervals is lower than that of the 30-40S subunits of completely dissociated 60-70S RNA; after

heating, their mobilities are identical. Heating also releases some small RNA's from 30-40S RNA of RSV harvested at 3-minute intervals, but five times more 4S RNA is released if the 30-40S RNA is allowed to convert to 60-70S in the virus. Association of the 30-40S RNA's with some RNA's of the 4-12S class may take place simultaneously with their conversion to 60-70S RNA.

- 4944 PATHOGENESIS OF MAREK'S DISEASE IN OLD CHICKENS: LESION REGRESSION AS THE BASIS FOR AGE-RELATED RESISTANCE. (E.) Sharma, J. M. (Reg. Poultry Res. Lab., East Lansing, Mich.), R. L. Witter and B. R. Burmester. *Infect Immun* 8(5):715-724, 1973.

Chickens of various age levels, free from prior infection, were simultaneously exposed to Marek's disease virus, and the response of each age group was recorded. Four- and 20-wk-old chickens of lines 15x7 and CM (commercial source) had substantial resistance to mortality and gross lesions. In contrast, in line 7, which was tested at 1-day, 2-, 4-, 8-, 12- and 16-wk age levels, 4-wk-old chickens were fully susceptible to clinical Marek's disease (MD), although resistance was demonstrated at 8-wk and older age levels. Genetically resistant chickens of line 6 maintained their resistance at all age levels tested. Pathogenesis of MD was compared in 12-wk-old and 1-day-old chickens of line 15x7. Within the 1-day-old group, 23% of the chickens died because of MD, whereas there were no deaths in the 12-wk-old group. Both groups developed viremia although duration, incidence, and levels of virus in the 1-day-old group were higher than in the 12-wk-old group. Although initially the 12-wk-old group responded by producing higher levels of antibody, the long term incidence of agar gel precipitin, immunofluorescent, and virus neutralization antibody in the two groups was similar. Gross and microscopic lesions of MD developed in both groups, but lesions regressed in the 12-wk-old group and persisted in the 1-day-old group. It is concluded that age resistance to MD is expressed through lesion regression.

- 4945 INDUCTION OF INTERFERON IN CHICK CELLS BY POLYOMA VIRUS. (E.) Ustacelebi, S. (Inst. Virology, Glasgow, Scotland) and J. F. Williams. *J Gen Virol* 21(1):163-168, 1973.

Polyoma virus induces high levels of interferon in non-permissive chick embryo fibroblast cultures, and certain features of this induction have been examined. In non-pre-treated cultures interferon first appears at around 30 hr after infection and reaches maximum levels at around 70 hr. An input multiplicity of at least 0.5 plaque forming units (p.f.u.)/cell is required for induction. In cultures pre-treated with interferon (primed), interferon induction is enhanced, with interferon first appearing in the medium at around 20 hr and reaching 4- to 8-fold higher levels than in non-primed cultures. In this case interferon is induced by an input multiplicity of as low as 0.1

p.f.u./cell. 'Full' or intact polyoma particles induce, but 'empty' shells do not. Interferon can also be induced by polyoma on continuously passaged lines of chick embryo fibroblasts. The findings support the view that either the nucleic acid or an internal protein of the virus is involved in induction.

- 4946 ELECTRON MICROSCOPE OBSERVATION OF VIRUS-LIKE PARTICLES IN COMEDOCARCINOMA OF THE HUMAN BREAST. (E.) Seman, G. (U. Texas, M. D. Anderson Hosp. Tumor Inst., Houston) and L. Dmochowski. *Cancer* 32(4):822-829, 1973.

Viruslike particles of two different types have been observed by electron microscope examination of a breast tumor specimen of a patient with comedocarcinoma and Hashimoto's disease. The particles have all been found in endoplasmic cisternae of epithelial tumor cells. Viruslike particles of one type, represented by 80-millimicron particles arranged in chainlike formations, showed some general features of the hamster type H virus particles; however, no distinct spokes originating from the nucleoid could be seen as described in type H particles. Viruslike particles of the other type resembled type C virus particles, but were larger (200 millimicrons) in size and, unlike type C particles, were found only in the cisternae of the endoplasmic reticulum. No viruslike particles have been found in biopsy specimens of two other patients with comedocarcinoma. It is not known whether the autoimmune disease diagnosed in the patient with comedocarcinoma of the breast has played any part in the finding of the viruslike particles, as other patients with similar histological type of breast cancer without symptoms of autoimmune disease have not revealed these viruslike particles.

- 4947 RNA-DNA COVALENT BONDS BETWEEN THE RNA PRIMERS AND THE DNA PRODUCTS FORMED BY RNA TUMOR VIRUS DNA POLYMERASE. (E.) Flugel, R. M. (Dept. Biochem., U. Wisconsin, Madison), U. Rapp and R. D. Wells. *J Virol* 12(3):1491-1502, 1973.

The initiation of DNA synthesis by endogenous RNA primer molecules was studied in three RNA tumor viruses (B77 strain of avian sarcoma virus (B77 virus), murine leukemia virus (MuLV), and avian myeloblastosis virus (AMV)). Ether disrupted virions of MuLV and B77 virus have rC-dC and rA-dA covalent linkages between RNA primers and newly synthesized DNA. None of the 14 other possible bonds were formed. Ether-disrupted virions of AMV have rU-dC and rA-dA linkages. In contrast, Nonidet P-40 (NP-40)-disrupted virions of all three viruses have only the rA-dA junction. Studies with virus particles which were first disrupted with ether and then treated with NP-40 indicated that the detergent treatment disallowed the formation of ribopyrimidine-dC internucleotide bond. The same transfers are found with AMV in the presence or absence of actinomycin D, where only single-stranded DNA is formed. This indicates that virtually all of the significant primers have been recognized.

Transfer experiments with ether-disrupted early harvest (5 min) MuLV showed only the rC-dC bond; the rA-dA bond was absent. The short-time harvest contains a significantly higher proportion of infectious virions than 24 hr harvests. Also, since the RNA from early harvest virus is appreciably more homogeneous than the RNA of mature MuLV, it appears that the ribopyrimidine-dC linkage is the more significant initiation event from a biochemical standpoint.

- 4948 COMPARISON OF SV40 DNA TRANSCRIPTION *IN VIVO* AND *IN VITRO*. (E.) Khoury, G. (Natl. Inst. Allergy Infectious Dis., Natl. Insts. Hlth., Bethesda, Md.) and M. A. Martin. *Nature [New Biol]* 238(79):4-6, 1972.

Hydroxyapatite chromatography was used to examine SV40-specific RNAs produced *in vitro* with E. coli RNA polymerase and *in vivo* during the course of lytic infection. *In vitro*, the SV40-specific RNA is transcribed from one strand of viral DNA. However, *in vivo*, portions of both viral DNA strands were copied, resulting in an RNA product that was complementary to about 50% of the SV40 genome. These data indicate that two distinct patterns of transcription are involved in the *in vivo* and *in vitro* production of SV40-specific RNAs by E. coli.

- 4949 VIRUS-LIKE PARTICLES IN AN EQUINE SARCOID CELL LINE. (E.) England, J. J. (Coll. Vet. Med. Biomed. Sci., Colorado State U., Fort Collins), R. E. Watson, Jr. and K. A. Larson. *Am J Vet Res* 34(12):1601-1603, 1973.

The Mcl equine sarcoïd cell line was examined under an electron microscope. Almost all the cells had virus-like particles in the cytoplasm, cytoplasmic vesicles, or extracellular spaces. Untreated Mcl cells and cells treated with dimethyl sulfoxide had 0 to 10 intracytoplasmic virus-like particles per cell, while idoxuridine-DMSO (IDU-DMSO)-treated cells had two to four times that number of particles per cell. Particles were also seen in the extracellular spaces of the IDU-DMSO-treated cells. The virus-like particles, most of which were free in the cytoplasm, were double membrane structures 80 to 100 nm in length and with electron-lucent cores of 60 nm. Budding particles were seen only at the plasma membrane. These particles were similar to certain oncornaviruses. The data indicate that the Mcl cells have sufficient genetic information for increased viral production.

- 4950 HERPESVIRUS CERVICITIS AND CERVICAL NEOPLASIA: A CYTOLOGIC REVIEW. (E.) Amstey, M. S. (U. Rochester Sch. Med., Dentistry, N.Y.), S. F. Patten and M. Turk. *Cancer* 32(6):1321-1324, 1973.

The cytologic data from 113 patients with a diagnosis of herpesvirus infection on Papanicolaou stained smears from the cervix were reviewed. Of these patients, 43 had additional cytologic abnormalities

ranging from atypical cells to carcinoma, and 28 had some form of cervical epithelial atypia before herpesvirus was found. The atypicalities observed in cervical smears prior to the demonstration of the presence of herpesvirus were generally more severe than those discovered after the diagnosis of herpesvirus. The smears of 86 patients showed morphological changes indicative of primary herpesvirus infection, while secondary-type changes were found in the smears of the remaining 27 patients. Fifty-eight patients with primary-type morphology and 12 patients with secondary-type morphology had no additional abnormalities. These data strongly suggest that cervical epithelial atypias precede primary herpesvirus infections.

- 4951 EFFECTS OF SIMIAN VIRUS 40-INDUCED TRANSFORMATION ON ISOACCEPTING SPECIES OF TRANSFER RNA FROM MOUSE FIBROBLASTS. (E.) Portugal, F. H. (Nat'l. Cancer Inst., Bethesda, Md.), J. S. Simonds, D. Twardzik and M. Oskarsson. *J Virol* 12(6):1616-1619, 1973.

The effects of simian virus 40 (SV40) transformation on the elution profiles of tRNA from embryonic BALB/c 3T3 mouse fibroblasts were studied. Quantitative differences for one or more isoaccepting species of tRNA were found for each of the amino acids (alanine, arginine, histidine, leucine, lysine, phenylalanine, serine, tyrosine, and valine) examined when tRNA from SV40-transformed cells was aminoacylated *in vitro* and co-chromatographed with tRNA prepared from newborn mice similarly acylated on benzoated DEAE-cellulose columns. The largest quantitative difference was found for isoaccepting species of lysyl-tRNA from SV40-transformed cells. The elution profiles for tRNA extracted from noninfected embryonic cells and murine sarcoma virus (MSV)-transformed cells were studied. Similar differences in the elution profiles of tRNA for the infected and noninfected cell lines were noted as was found for tRNA extracted from SV40-transformed cells. The data suggest that when utilizing normal mouse cell aminoacyl synthetases, no unique isoaccepting tRNA species for alanyl-, arginyl-, histidyl-, leucyl-, lysyl-, phenylalanyl-, seryl-, tyrosyl-, and valyl-tRNAs in cells transformed by SV40 can be detected. Significant differences between infected cells in culture and newborn mouse cells are attributed to tRNA alterations accompanying the differentiation of mouse cells.

- 4952 DNA POLYMERASE OF MURINE SARCOMA-LEUKEMIA VIRUS: LACK OF DETECTABLE RNase H AND LOW ACTIVITY WITH VIRAL RNA AND NATURAL DNA TEMPLATES. (E.) Wang, L.-H. (Dept. Molecular Biol., U. California, Berkeley) and P. H. Duesberg. *J Virol* 12(6):1512-1521, 1973.

Kirsten murine sarcoma-leukemia virus (Ki-MSV(MLV)) was found to contain less RNase H per unit of viral DNA polymerase than avian Rous sarcoma virus (RSV). Upon purification by chromatography on Sephadex G-200 and subsequent glycerol gradient sedimentation, the avian DNA polymerase was obtained in

association with a constant amount of RNase H. In contrast, equally purified DNA polymerase of Ki-MSV(MLV) and Moloney mouse leukemia virus (Mo-MSV(MLV)) lacked detectable RNase H if assayed with two homopolymer and phage *fd* DNA-RNA hybrids as substrates. On the basis of picomoles of nucleotides turned over, the ratio of RNase H to purified avian DNA polymerase was 1:20 and that of RNase H to purified murine DNA polymerase ranged between less than 1:2800 and 5000. Based on the same activity with poly (A)·oligo (dT), the activity of the murine DNA polymerase was 6 to 60 times lower than that of the avian enzyme with denatured salmon DNA template or with avian or murine viral RNA templates assayed under various conditions (native, heat-dissociated, with or without oligo(dT) and oligo(dC), and at different template enzyme ratios). The template activities of Ki-MSV(MLV) RNA and RSV RNA were enhanced uniformly by oligo(dT), but oligo(dC) was much less efficient in enhancing the activity of MSV(MLV) RNA than that of RSV RNA. The purified DNA polymerase of Ki-MSV(MLV) appears to differ from that of RSV in its lack of detectable RNase H and in its low capacity for transcribing viral RNA and denatured salmon RNA.

- 4953 BIOLOGICAL AND BIOCHEMICAL EVIDENCE FOR AN INTERACTION BETWEEN MAREK'S DISEASE HERPESVIRUS AND AVIAN LEUKOSIS VIRUS *IN VIVO*. (E.) Peters, W. P. (Inst. Cancer Res., Coll. Physicians, Surgeons, Columbia U., New York, N.Y.), D. Kufe, J. Schlom, J. W. Frankel, C. O. Prickett, V. Groupé and S. Spiegelman. *Proc Nat Acad Sci* 70(11):3175-3178, 1973.

Three-day old S-line and LSI-SPF chickens were inoculated interperitoneally with Marek's disease herpesvirus (MDHV) or Rous-associated virus type 2 (RAV-2); at bimonthly intervals thereafter, similarly inoculated and uninoculated chicks were placed in the same housing facility. None of the uninoculated LSI-SPF chicks exposed to either MDHV or RAV-2 died or showed any evidence of tumors during the 60 day observation period; however, uninoculated LSI-SPF chicks exposed to both MDHV and RAV-2 showed an 81% mortality rate and a 100% incidence of tumor development. Gross microscopic examination of the tissues of the affected birds revealed the massive visceral and neural lymphocytic infiltration characteristic of Marek's disease. In addition, there was an 88% rate of mortality among S-line chickens exposed to both viruses, as compared to the 18% rate among those exposed only to MDHV and the 0% rate among those exposed only to RAV-2. Gross and microscopic lesions characteristic of Marek's disease were observed in the liver, kidney, spleen, and thymus from all chickens showing Marek's disease symptomatology. Molecular hybridization data indicated a significantly higher level of Rous sarcoma leukosis virus specific RNA in the tissues of birds exposed to MDHV and RAV-2 simultaneously or to MDHV alone than was observed in the corresponding tissues of unexposed birds or birds contact-exposed to RAV-2 alone. Thus, under the conditions of these experiments, an interaction occurs between Marek's disease herpesvirus and an avian leukosis virus *in vivo*.

4954 SPONTANEOUS PRODUCTION OF A C-TYPE RNA VIRUS IN RAT TISSUE CULTURE LINES. (E.)

Klement, V. (U. Southern California Sch. Med., Los Angeles), M. O. Nicolson, W. Nelson-Rees, R. V. Gilden, S. Oroszlan, R. W. Rongey and M. B. Gardner. *Int J Cancer* 12(3):654-666, 1973.

The induction of a C-type RNA virus (XCIV) in tissue culture line XC derived from Wistar random-bred rats was recently demonstrated. Two other rat C-type virus isolates (NRKAV and RPLAV) are herein described. They were associated with and isolated from, NRK and RPL lines originating in Osborne-Mendel and Lewis rat strains, respectively. The main biological characteristics shared by the three viruses were buoyant densities in sucrose gradients in the range of 1.14 to 1.15 g/ml, endogenous DNA polymerase activity, high molecular weight (60 to 70S) RNA, rat major species-specific (gs-1) antigen and gs-3 interspecies antigen. Two of the isolates (NRKAV and RPLAV) were able to rescue mouse sarcoma virus (MSV) genome from a rat MSV-transformed non-productive line. The NRK line was assayed for virus release at various *in vitro* passage levels. In lower passages (43-73), spontaneous virus release was absent or minimal although the virus could be induced by treatment of the cells with 5-bromodeoxyuridine. At higher passage levels, the cells produced a large amount of virus spontaneously, but in these treatment with 5-bromodeoxyuridine decreased virus production. The results of karyological analysis suggested that clonal selection was taking place in the NRK line during consecutive *in vitro* passages and that it occurred in conjunction with the process of increasing spontaneous virus release. Thus, the mechanism of the cellular regulation of complete type-C virus synthesis includes functions other than those involved in transformation.

4955 EARLY SYNTHESIS OF VIRUS-SPECIFIC RNA AND DNA IN CELLS RAPIDLY TRANSFORMED WITH ROUS SARCOMA VIRUS. (E.) Schincariol, A. L. (Duke U. Med. Ctr., Durham, N.C.) and W. K. Joklik. *Virology* 56(2):532-548, 1973.

Chick embryo fibroblasts (CEF) were infected with the Prague strain (subgroup C) of Rous sarcoma virus (PrC-RSV) under conditions of rapid transformation, and the synthesis of RNA and DNA capable of hybridizing with single-stranded DNA transcripts of 70S PrC-RSV RNA or DNA was measured via hybridization kinetics analysis. PrC-RSV DNA was synthesized in the presence of high concentrations of actinomycin D and was freed from double-stranded DNA by chromatography; it protected over 90% of 70S PrC-RSV RNA from digestion by ribonuclease at DNA:RNA ratios greater than 9. Uninfected gs-CEF contained on the average one viral genome copy of DNA per cell and no detectable virus-specific RNA. DNA capable of hybridizing with the probe began to be synthesized within 3 hr after infection, and by 24 hr, two viral genome copies had been synthesized. No additional virus-specific DNA was synthesized thereafter. During the first 6 hr post-infection, CEF contained about 40 viral genome equivalents of RNA per cell, about 85% of which were in the cytoplasm.

This probably represented RNA in inoculum virus. Between 6 and 24 hr, the number of viral genome equivalents per cell increased to 100. This newly synthesized RNA was equally distributed between the nucleus and polyribosomes. Between 24 and 96 hr, the amount of virus-specific RNA in the nucleus and polyribosomes increased by just under 2-fold, although in the remainder of the cytoplasm it rose by over 8-fold. By 96 hr, the cells contained about 340 genome equivalents of RNA per cell, which was about 1/2 of the number present in the cells 14 days post-infection. The size of the virus-specific RNA present in the polyribosomes was predominantly 35 S, but some 10 to 30S material was also present.

4956 FORMATION OF LIPID-NUCLEOTIDE COMPLEX BY RNA TUMOR VIRUS PREPARATIONS. (E.) Flugel, R. M. (Dept. Biochem., U. Wisconsin, Madison) and R. D. Wells. *J Virol* 12(6):1622-1624, 1973.

During an investigation into the effect of disruption conditions on the RNA-DNA covalent linkages formed by the endogenous reaction with three RNA tumor viruses (avian myeloblastosis virus, B77 virus, and murine leukemia virus), it was found that a portion of the labeled deoxyribonucleoside triphosphate substrates were being converted into a complex nucleotide-lipid product which was tightly associated with protein. This product, which constituted 0.5 to 3% of the DNA formed, was formed by either NP-40- or ether-disrupted preparations of all RNA tumor viruses studied; this was roughly the extent of formation of RNA-DNA linkages. The product was found to contain phospholipid, protein, and dNMP, as well as a nucleotide.

4957 RIBOSOMES FROM RAUSCHER LEUKEMIA VIRUS-INFECTED CELLS AND THEIR RESPONSE TO RAUSCHER VIRAL RNA AND POLYURIDYLIC ACID. (E.) Naso, R. B. (Dept. Biol., U. Texas, Houston), C. S. Wang, S. Tsai and R. B. Arlinghaus. *Biochem Biophys Acta* 324(3):346-364, 1973.

A detailed description of the methods used to prepare polyribosomes, ribosomes, and ribosomal subunits from Rauscher leukemia virus (RLV) infected cells derived from the spleen and thymus of weanling BALB/c mice is presented. The various ribosome preparations were analyzed by rate-zonal centrifugation on sucrose gradients, assayed for endogenous polypeptide synthesis and tested for their response to polyuridylic acid (poly(U)) and RLV-RNA. A " Mg^{2+} -shift" assay for protein synthesis initiation factors was established in this system. The 65-S 3H -labeled RLV-RNA binds to ribosomes and forms dimeric through tetrameric polyribosome-like structures in a cell-free system. The ribosome binding requires purified ribosomal subunits, GTP, tRNA, and 0.5 M KCl wash of whole cell polyribosomes, and can be accomplished under conditions where no acid precipitable polypeptides are formed. The 0.5 M KCl wash was active in the " Mg^{2+} -shift" assay with poly(U) indicating that it contains initiation factors M_1 and M_2 . The formation of 3H -labeled RLV-RNA polyribosomes was indicated by their density in CsCl gradients after

glutaraldehyde fixation, their binding to cellulose nitrate filters, and their sedimentation properties in sucrose gradients. Heat-dissociated 65-S ^3H -labeled RLV-RNA (35 S and lower s forms) formed mainly monosomes. The protein product made in response to 65-S RLV-RNA in preincubated 30-S extracts contained two high molecular weight polypeptides estimated at 185,000 and 125,000. All of these data taken together lead to several possible conclusions: 1. Each 65-S RLV-RNA has three to four ribosomal binding sites; 2. Each 35-S RLV-RNA subunit contains one ribosomal binding site; 3. The 65-S RLV-RNA is translated as a large polypeptide which could be analogous to the precursor "polyprotein" seen in picornavirus-infected cells.

- 4958 LOW-MOLECULAR-WEIGHT RNAs OF MURINE SARCOMA VIRUS: COMPARATIVE STUDIES OF FREE AND 70S RNA-ASSOCIATED COMPONENTS. (E.) Emanoil-Ravicovitch, R. (Leukemia Res. Inst., Paris, France), C. J. Larsen, M. Bazilier, J. Robin, J. Peries and M. Boiron. *J Virol* 12(6):1625-1627, 1973.

Moloney strain mouse sarcoma virus were prepared from growth fluids of 78 A₁, a chronically infected cell line, cultured, and labeled with ^{32}P - or ^3H -uridine. The cell supernatants were harvested 18 hr later, and RNA extracted from the purified virus. The 70S RNA was then subjected to heat denaturation, and its components separated by polyacrylamide-agarose gel electrophoresis. Several low-molecular-weight RNAs were thus obtained, one of which exhibited the same mobility as the free viral 8S RNA previously described in this virus. The 70S-associated 8S RNA also presented the same nucleotide composition as the free viral 8S RNA. In addition to the 8S species, 4S, 5S, and 5.5S RNAs were separated from heat denatured 70S RNA. These data indicate a close relationship between murine and avian oncoviruses in their ability to release light RNA components by denaturation of the 70S RNA; the presence of 4S RNA seems to be a common feature of these viruses, although 70S-associated 8S RNA is not known to be present in the avian viruses.

- 4959 ANALYSIS OF MINIMAL FUNCTIONS OF SIMIAN VIRUS 40. II. ENHANCEMENT OF ONCOGENIC TRANSFORMATION *IN VITRO* BY UV IRRADIATION. (E.) Seemayer, N. H. (Wistar Inst. Anatomy, Biol., Philadelphia, Pa.) and V. Defendi. *J Virol* 12(6):1265-1271, 1973.

Syrian hamster kidney cell cultures were infected with light UV irradiated (5000 to 10,000 ergs/mm²) "complete" and "defective" simian virus 40 (SV40). The irradiation increased the oncogenic transformation capacity of the complete pool by up to 180% and that of the defective pool by up to 270%. There was a concomitant increase in the T-antigen induction of CV-1 cells infected with irradiated SV40; however, infectivity was reduced by 1 log₁₀. After strong UV irradiation (10,000 to 60,000 ergs/sq mm) the transformation capacity of the complete and defective SV40 pools was reduced to 30 to 70% that of nonirradiated pools; infectivity was reduced by

more than 3.5 log₁₀. With additional irradiation, the rate of decrease of the transformation capacity was more rapid, even though at a dose of 80,000 ergs/sq mm, 2 to 5% of the transformation capacity remained; infectivity was reduced by 4 to 5 log₁₀, resp., for the complete and defective pools. T-antigen induction of SV40 was equally resistant to strong UV irradiation. No evidence was found of an involvement of "multiplicity reactivation" in the high resistance of the transformation capacity of SV40 following UV irradiation. Syrian hamster kidney cells transformed *in vitro* by UV-irradiated SV40 contained the SV40-specific T-antigen and showed the same morphology and growth characteristics as cells transformed by nonirradiated complete or defective SV40. They induced malignant tumors following s.c. inoculation into Syrian hamsters.

- 4960 AMINOACYL-tRNA SYNTHETASE ACTIVITY IN VIRIONS OF AVIAN MYELOBLASTOSIS VIRUS. (E.) Travnickek, M. (Inst. Organic Chem., Biochem., Czechoslovak Acad. Sci., Prague) and J. Riman. *Neoplasma* 20(2):113-123, 1973.

Purified BAI strain A avian myeloblastosis virus (AMV), obtained from the heparinized blood plasma of leukemic chicks, was centrifuged and assayed for aminoacyl-tRNA synthetase activity. The aminoacyl-tRNA synthetase activity of purified leukemic myeloblasts from the fresh heparinized blood of leukemic chicks was also determined. The native virions exhibited no aminoacylating activity and inhibited the aminoacylation of tRNA catalyzed by the host enzyme; this inhibitory effect was due to ATPase activity associated with the outer membrane of the native AMV. When AMV virions pretreated with the nonionic detergent Nonidet NP-40 were assayed for enzyme activity, aminoacyl-tRNA synthetase activity was demonstrated. Based on the observation that the specific enzyme activity did not change as a result of further extensive virus purification, it was concluded that aminoacyl-tRNA synthetase activity is present within the AMV virions. Lysyl-tRNA synthetase prevailed in AMV virions in accordance with the previous finding of high levels of tRNA^{Lys}. Viral aminoacyl-tRNA synthetase was purified by DEAE-cellulose column chromatography and some of its basic characteristics determined. The aminoacyl-tRNA synthetase reaction catalyzed by the enzyme isolated from AMV virions was proportional to the amount of enzyme added and dependent on added cellular tRNA; the reaction was linear with time. The virus-occluded protein-synthesizing system may play a role in the virus-infected cells or directly in the virions, or it may be a nonfunctional feature of animal viruses.

- 4961 DETECTION OF VIRAL DNA SEQUENCES IN ADENOVIRUS-TRANSFORMED CELLS BY *IN SITU* HYBRIDIZATION. (E.) Loni, M. C. (St. Louis U. Sch. Med., Mo.) and M. Green. *J Virol* 12(6):1288-1292, 1973.

Cytological preparations of rat and hamster cells transformed by members of three groups of human

adenoviruses (adenovirus 12, 7 and 2) were annealed with radioactive complementary RNA (40,000,000 to 45,000,000 dpm/ μ g) prepared by copying viral DNA with the *Escherichia coli* DNA-directed RNA polymerase. These *in situ* hybridizations detected adenovirus-specific DNA sequences in interphase nuclei when transformed cells were annealed with homologous viral cRNA, but not with heterologous viral cRNA. The highest autoradiographic grain counts were found over adenovirus 7-transformed cell nuclei (3.31 grains per nucleus); the next highest were found over adenovirus 12-transformed nuclei (1.72 grains per nucleus); and the lowest were found for adenovirus 2-transformed cell nuclei (0.85 grains per nucleus). The estimated genome equivalent per cell is 10.7, 5.5, and 2.7, resp., for adenovirus 7-, 12-, and 2-transformed cells. This is the same order that was found by reassociation kinetic measurements.

- 4962 NUCLEIC ACID SYNTHESIS IN CYTOPLASM OF YABA MONKEY TUMOR VIRUS-INFECTED CELLS. (E.) Rouhandeh, H. (Dept. Microbiol., Southern Illinois U., Carbondale) and M. L. Rouhandeh. *J Virol* 12(6):1407-1413, 1973.

The synthesis of DNA in Yaba virus-infected cells was monitored via the incorporation of acid-insoluble radioactive thymidine into the cytoplasmic fraction of established cynomolgus monkey kidney cells. Yaba tumor virus progeny appeared in the kidney cells at 24 hr postinfection and reached a plateau at 72 hr in the first cycle of replication. Viral DNA synthesis was first detected at about 3 hr and reached a peak after 18 hr. The maximum coating of the viral DNA in the infected cells occurred at 4 days postinfection. Rapidly labeled RNA was synthesized in the cytoplasm of the virus-infected cells. At 6 hr postinfection, 7 to 10S RNA was present; this species was present in greater quantities at 12 hr; at 24 hr, a truncated peak indicated the presence of 14 to 15S as well as 7 to 10S RNA. Hybridization data indicated that the largest peak of messenger RNA synthesis occurred at 11 to 13 hr postinfection, and that a second, slightly smaller, peak occurred at 21 to 23 hr. Although the time course of Yaba virus DNA synthesis follows a pattern similar to that reported for other poxviruses, the entire pattern is considerably slower.

- 4963 RIBOSOMAL RNA SYNTHESIS AFTER INFECTION WITH ADENOVIRUS TYPE 2. (E.) Eliceiri, G. L. (St. Louis U. Sch. Med., Mo.). *Virology* 56(2):604-607, 1973.

Cultures of mouse parental cells (3T3-4E), hamster parental cells (T6a), and hamster-mouse hybrids were infected with adenovirus type 2 (Ad 2) preparations, and their ribosomal RNA synthesis studied. Ad 2-infected T6a cells underwent lysis, while infected 3T3-4E cells did not. Similarly, while there was a marked decrease in the incorporation of (3H) uridine into rRNA in the Ad 2-infected T6a cells, there was no such decrease in the 3T3-4E cells; in comparison with the 4S RNA of the T6a cells, the

incorporation of (3H)uridine into rRNA was specifically inhibited. This indicates that the synthesis of mature cytoplasmic rRNA was inhibited, rather than the ribonucleotide pool. No preferential leakage of 4S RNA from the infected cells occurred. Mature cytoplasmic rRNA was also inhibited in the mouse-hamster hybrids with a majority of hamster chromosomes. No significant change in the proportion of hamster and mouse cytoplasmic ribosomal RNA synthesized was detected. Thus, once the lytic cycle is started, the synthesis of both hamster and mouse rRNA appears to be equally affected in these hybrids, with no preferential effect.

- 4964 INFLUENCE OF AGE AT EXPOSURE ON THE PATHOGENESIS OF MAREK'S DISEASE. (E.) Calnek, B. W. (New York State Vet. Coll., Cornell U., Ithaca). *J Natl Cancer Inst* 51(3):929-939, 1973.

In seven experiments, 1-day- to 4½-month-old chickens, free of Marek's disease virus (MDV) and MDV antibody, were exposed to clone-purified virus. Cellular preparations of the GA or JM isolate of MDV were inoculated intra-abdominally, or birds were exposed to GA virus by indirect contact. Genetically susceptible P-line birds were compared to those of the genetically resistant N-line and relatively resistant PDRC strain. Response to infection in various experiments was assessed by: incidence of MD through 56 or 70 days; infectivity of buffy coat, spleen, and kidney cells; presence of viral antigen (fluorescent antibody test) in bursa of Fabricius, spleen, and thymus; and development of precipitin and virus-neutralizing (VN) antibody. Age resistance was characterized by a) lower incidence of MD, b) reduced rate and level of infection 7-10 days post inoculation, and c) development of VN antibody. A strong age resistance developed consistently in the N-line and PDRC birds but was weak or absent in the P-line. Resistance was acquired gradually during several weeks and paralleled the acquisition of competence to make precipitin or VN antibody. During the first 3-6 days after inoculation, infection of lymphoid organs was marked and proceeded equally in all groups, regardless of age or genetic strain. However, by 10-12 days post infection, the level of infection subsided in resistant birds but not in those susceptible.

- 4965 LOCALIZATION OF RNA TUMOR VIRUS POLYPEPTIDES. I. ISOLATION OF FURTHER VIRUS SUBSTRUCTURES. (E.) Bolognesi, D. P. (Duke U. Med. Ctr., Durham, N. C.), R. Luftig and J. H. Shaper. *Virology* 56(2):549-564, 1973.

The internal components of avian myeloblastosis virus (AMV) and Friend murine leukemia virus (FLV) were isolated and characterized. For both of these viruses, most of the major structural proteins were constituents of the isolated cores. The AMV cores contained three of the five major virus polypeptides; they had molecular weights of 27,000, 15,000, and 12,000. The FLV cores contained three of the four major components, these having molecular weights of 31,000, 15,000, and 10,000. The 15,000 dalton component was

present in the smallest amounts in both virus cores. The internal ribonucleoprotein was isolated from each of the cores and was shown to consist of the most basic protein of the respective agents. The properties and locations of the proteins of both viruses were similar although the structural characteristics of the avian and mammalian cores appeared to be somewhat different.

- 4966 GROWTH INDUCTION BY SERUM OR POLYOMA VIRUS INHIBITS THE AGGREGATION OF TRYPSIN-ISED SUSPENSIONS OF BHK21 TISSUE CULTURE FIBROBLASTS. (E.) O'Neill, C. H. (Imperial Cancer Res. Fund Lab., London, England). *Exp Cell Res* 81:31-39, 1973.

BHK21 hamster tissue culture fibroblasts, when brought into suspension with trypsin, aggregate spontaneously in serum-free medium. The amount of aggregation appears to depend on the proportion of quiescent (growth inhibited) cells which were present in the culture. It is greatest in cells from cultures whose growth is inhibited, either by high cell density or by low serum concentration. When serum is added to low serum cultures, growth is induced and S phase and mitosis follow at 14 and 20 hr resp. It has now been found that aggregation ceases less than 2 hr after the addition of serum. Growth can also be induced in such cultures by infection with high multiplicities of polyoma virus. Infected cells enter S phase about 28 hr after infection. It has been found that aggregation ceases between 12 and 20 hr after infection. Thus in both these cases loss of aggregation precedes S phase by about 12 hr. It is concluded that the absence of aggregation in suspensions derived from polyoma virus-transformed lines of BHK21 cells is a consequence of their resistance to density-dependent inhibition of growth. Failure to undergo spontaneous aggregation appears to be an indicator of an early surface change associated with the induction of growth.

- 4967 ROLE OF HERPES HOMINIS 2 VIRUS IN THE GENESIS OF CERVICAL CANCER. (Ger.) Horacek, J. (Microbiol. Inst., Charles U., Prague, Czechoslovakia) and M. Rosol. *Z Gynaekol* 95(36): 1278-1281, 1973.

Attempts were made to isolate Herpes hominis 2 virus (HVH-2) from 70 women who were suspected of having cervical carcinoma on the basis of cytological examinations. These women consisted of 8 with invasion squamous cell carcinomas, 2 with invasive adenocarcinomas, 3 with Mestwerdt microcarcinomas, 13 with carcinoma *in situ*, 18 with cellular atypia, and 12 with epithelial dysplasia; 14 patients were later found to have false-positive cytology. Repeat attempts at isolation were made after delivery in 5 pregnant women. In each case material from the cervix was inoculated into monkey kidney cells, human embryonic fibroblasts, and HeLa cells. No cytopathic effect was observed in any of the 65 cases that could be evaluated. In 5 cases the results could not be evaluated because of contamination with bacteria or fungi or toxic damage to

the cells. HVH-2 was isolated by the same technique from two men with herpes progenitalis. It is possible that high neutralization antibody titers for HVH-2 previously found in women with cervical cancer or precancer are only a sign that these women have had more sexual contacts.

- 4968 A DIFFERENCE IN THE BREAKDOWN OF PHOSPHATIDYLINOSITOL IN NORMAL AND SV40 TRANSFORMED MOUSE FIBROBLASTS. (E.) Koch, M. A. (Max-Planck Inst. Virol., Tübingen, W. Germany) and H. Diringer. *Biochem Biophys Res Commun* 55(2):305-311, 1973.

Phospholipid metabolism was studied in exponentially growing and contact-inhibited SV40-transformed mouse fibroblasts (STU-261) pulse-chased with ^{32}P -orthophosphate and ^3H -glycerol. A prolonged uptake of radioactivity into phosphatidylethanolamine compared to phosphatidylcholine and phosphatidylinositol was observed in exponentially growing cells but not in high density cultures produced by addition of unlabeled cells to the culture. The rate of loss of ^{32}P from phosphatidylcholine and phosphatidylethanolamine was slower in high density than in exponentially growing cultures. However, no difference was observed between high and low density cultures in the rate of phosphatidylinositol breakdown. In addition, the $^3\text{H}:^{32}\text{P}$ ratio in phosphatidylinositol increased steadily only in exponentially growing cultures, indicating a lack of reutilization of the glycerol moiety in high density cultures. The only difference observed between the transformed STU-261 cells and normal mouse fibroblasts was that high density normal fibroblast cultures did show a three-fold decrease in the rate of loss of ^{32}P from phosphatidylinositol.

- 4969 REPLICATION OF ADENOVIRUS 12 DEOXYRIBONUCLEIC ACID IN ASSOCIATION WITH THE NUCLEAR MEMBRANE. (E.) Yamashita, T. (Nat'l. Inst. Hlth., Musashimurayama, Japan) and H. Shimajo. *Jap J Microbiol* 17(5):419-423, 1973.

Analysis of nuclei of adenovirus 12-infected human embryo kidney cells revealed that viral DNA replicated in association with the nuclear membrane and that complete viral DNA was liberated from the nuclear membrane. Examination of isolated nuclei *in vitro* showed that DNA polymerase activity increased in the nuclear membrane of adenovirus 12-infected cells without addition of primer DNA. It is suggested that the complete machinery for adenovirus DNA replication consists of many factors, such as parental viral genome, components of the nuclear membrane, cellular and viral proteins synthesized *de novo* for viral DNA replication and so on.

- 4970 TUMOR PRODUCING CAPACITY OF TEMPERATURE SENSITIVE MUTANTS OF AVIAN SARCOMA VIRUSES IN CHICKS. (E.) Toyoshima, K. (Res. Inst. Microbial Dis., Osaka U., Japan), M. Owada and Y. Kozai. *Biken J* 16(3):103-110, 1973.

Tumor production by five temperature sensitive (ts)

mutants of avian sarcoma virus was tested to examine the relation between temperature sensitivity of cell transformation *in vitro* and the tumor-producing capacity *in vivo*. When ts mutants were inoculated into the wing webs of chicks, the incidence of tumors was low. However, when the inoculated chicks were exposed to unfavorable conditions during the first wk of infection, which lowered the temperature of the inoculated site, tumors appeared at high frequencies and grew rapidly. These results suggest that mutants which possess ts characters of cell transformation *in vitro* also have a tumor-producing capacity *in vivo* which is affected by temperature. Some, though not all, of the tumors produced *in vivo* by ts mutants contained back mutants.

- 4971 A POLYOMA-INDUCED HAMSTER TUMOR CELL WITH ALTERED DNA SYNTHESIS *IN VIVO* AND REDUCED POLYNUCLEOTIDE LIGASE ACTIVITY *IN VITRO*. (E.) Coscia, J. F., Jr. (U. Rochester Sch. Med. Dentistry, N.Y.) and J. D. Hare. *Physiol Chem Phys* 5(4):271-285, 1973.

An abnormality in the DNA replication mechanism of a polyoma-induced hamster tumor cell line which reverts to normal on continuous passage in culture was studied. A distinctly different pattern of distribution of newly replicated DNA was observed in the tumor cell line as compared to normal embryo cells when sonicated cell lysates were analyzed on an alkaline CsCl gradient following a 10 min pulse with ³H-thymidine (dT) and a chase with bromodeoxyuridine (BrdU). A significant proportion of the newly replicated, ³H-labeled DNA in the tumor cell shifted to a high density compatible with incorporation of BrdU into replicating DNA fragments that did not become covalently linked to light DNA. When polynucleotide ligase activity was measured, a significant reduction in ligating activity of early passage tumor cell homogenates was demonstrated, supporting the hypothesis that the slow conversion of low molecular wt DNA pieces to high molecular wt native DNA is due to a defect in the ligation step in a discontinuous DNA synthesis mechanism.

- 4972 A QUANTITATIVE DIFFERENCE IN THE MOVEMENT OF MARKER PARTICLES IN THE PLASMA MEMBRANE OF 3T3 MOUSE FIBROBLASTS AND THEIR POLYOMA TRANSFORMANTS. (E.) Albrecht-Bühler, G. (Friedrich Miescher Inst., Basel, Switzerland). *Exp Cell Res* 78:67-70, 1973.

- 4973 STUDIES OF GENETIC TRANSMISSION OF MURINE LEUKEMIA VIRUS BY AKR MICE. II. CROSSES WITH Fv-1^b STRAINS OF MICE. (E.) Rowe, W. P. (Nat'l. Inst. Allergy and Infectious Dis., Bethesda, Md.) and J. W. Hartley. *J Exp Med* 136(5):1286-1301, 1972.

- 4974 RAPID METHOD FOR THE CONCENTRATION AND PARTIAL PURIFICATION OF HERPES SIMPLEX VIRUSES TYPES 1 AND 2. (E.) Lancz, G. J. (Coll. Med., U. South Florida, Tampa). *Arch Gesamte Virusforsch* 42(3):303-306, 1973.

- 4975 EXPERIMENTAL INFECTION WITH MOUSE ADENOVIRUS IN ADULT MICE. (E.) Van Der Veen, J. (St. Elisabeth Hosp., Tilburg, The Netherlands) and A. Mes. *Arch Gesamte Virusforsch* 42(3):235-241, 1973.

- 4976 STUDIES ON GROUP-SPECIFIC DETERMINANTS OF ADENOVIRUS HEXONS. (E.) Döhner, L. (Med. Acad., Magdeburg, E. Germany), U. Dieckmann and H. Struy. *Arch Gesamte Virusforsch* 42(3):254-263, 1973.

- 4977 DNA-DEPENDENT DNA POLYMERASE PATTERN IN NONINFECTED AND HERPESVIRUS INFECTED RABBIT KIDNEY CELLS. (E.) Müller, W. E. G. (Inst. Physiol. Chem., U. Gutenberg, Mainz/Rhein, Germany), D. Falke and R. K. Zahn. *Arch Gesamte Virusforsch* 42(3):278-284, 1973.

- 4978 GENETIC STUDIES ON HERPES SIMPLEX VIRUS. I. RECOMBINATION BETWEEN PLAQUE MORPHOLOGY AND IDU (5-iodo-2'-deoxyuridine) RESISTANCE. (E.) Yamamoto, S. (Kurume U. Sch. Med., Japan) and H. Kabuta. *Kurume Med J* 20(2):87-93, 1973.

- 4979 FORMATION OF ANUCLEATE AND MULTINUCLEATE CELLS IN NORMAL AND SV₄₀ TRANSFORMED WI-38 BY CYTOCHALASIN B. (E.) Wright, W. E. (Stanford U. Sch. Med., Calif.) and L. Hayflick. *Exp Cell Res* 74:187-194, 1972.

- 4980 CONCURRENT LYMPHOCYTIC LYMPHOMA AND INFECTIOUS MONONUCLEOSIS. (E.) Weiss, R. B. (West Virginia Univ. Hosp., Morgantown) and B. J. Kennedy. *Minn Med* 56(11):958-959, 1973.

- 4981 IMMUNOFLUORESCENCE AND ELECTRON MICROSCOPY OF THREE LYMPHOBLASTOID LINES WHICH ACT AS CARRIERS OF EPSTEIN-BARR TYPE HERPES VIRUS. (Fr.) Dalens, M. (Med. Sci. Res. Unit, Paul Sabatier U., Toulouse, France), J. Didier and L. Enjalbert. *C R Sci Soc Biol (Paris)* 167(3/4):576-581, 1973.

- 4982 NEUROLOGICAL COMPLICATIONS OF BURKITT'S DISEASE. CLINICAL AND BIOLOGICAL STUDY. (Fr.) Bonhomme, J. S. (No affiliation), J. P. Bureau and T. Schmitt. *Now Rev Fr Hematol* 13(5):681-688, 1973.

- 4983 POLYOMA VIRUS INHIBITORS. I. *IN VITRO* EXPERIMENTS. (Ger.) Desselberger, U. (Med. Coll., Hannover, Germany). *Zentralbl Bakteriol [Orig A]* 224(4):438-447, 1973.

- 4984 PROVISIONAL LABELS FOR HERPESVIRUS. (E.)
Internat'l Committee Nomenclature of
Viruses. *J Gen Virol* 20(3):417-419, 1973.
- 4985 DISRUPTION OF HERPES VIRUS NUCLEOCAPSIDS
USING LITHIUM IODIDE, GUANIDINE AND MER-
CAPTOETHANOL. (E.) McCombs, R. M. (Baylor Coll.
Med., Houston, Tex.) and G. A. Williams. *J Gen
Virol* 20(3):395-400, 1973.
- 4986 CONCENTRATION OF EPSTEIN-BARR VIRUS FROM
CELL CULTURE FLUIDS WITH POLYETHYLENE
GLYCOL. (E.) Adams, A. (Karolinska Inst., Stock-
holm, Sweden). *J Gen Virol* 20(3):391-394, 1973.
- 4987 RESTORATION OF THE FUSION ACTIVITY OF L
CELL-BORNE SENDAI VIRUS BY TRYPSIN. (E.)
Homma, M. (Tohoku U. Sch. Med., Sendai, Japan) and
J. Tomogau. *J Gen Virol* 1973(19):423-426, 1973.
- 4988 VIRUS-INDUCED CHANGES IN THE ENDOGENOUS
PATHWAY OF THYMIDINE IN ANIMAL CELLS.
(E.) Fuchs, P. (Ness-Ziona and Tel Aviv U. Med.
Sch., Romat Aviv, Israel) and A. Kohn. *Isr J Med
Sci* 9(4):477-480, 1973.
- 4989 ADENYLATE CYCLASE AND CYCLIC AMP PHOSPHO-
DIESTERASE OF NORMAL AND ROUS SARCOMA VIRUS
TRANSFORMED CHICKEN EMBRYO FIBROBLASTS. (E.)
Russell, T. R. (Natl. Cancer Inst., Bethesda, Md.)
and W. B. Anderson. *J Supramol Struct* 1(4/5):382-384,
1973.
- 4990 BIOSYNTHESIS OF VIRAL NUCLEIC ACIDS.
(Rus.) Agabalian, A. S. (Inst. Exp. Biol.,
Armenian Acad. Sci., USSR). *Biol Zh Armenii* (5):
31-38, 1973.
- 4991 ROLE OF CELLULAR REPARATIVE MECHANISMS IN
THE DEVELOPMENT OF SPONTANEOUS AND INDUCED
MUTATIONS IN MAMMALIAN VIRUSES. (Rus.) Zasukhina,
G. D. (Inst. Poliomyelitis Viral Encephalitis, Moscow,
USSR), V. A. Nesmashnova and G. N. L'vova. *Dokl
Akad Nauk SSSR* 212(1):223-225, 1973.
- 4992 STUDIES OF GENETIC TRANSMISSION OF MURINE
LEUKEMIA VIRUS BY AKR MICE. I. CROSSES
WITH Fv-1ⁿ STRAINS OF MICE. (E.) Rowe, W. P. (Natl.
Inst. Allergy and Infectious Dis., Bethesda, Md.).
J Exp Med 136(5):1272-1285, 1972.

See also:

- * (Rev): 4806, 4808
- * (Chem): 4854, 4865
- * (Immun): 4995, 5000, 5005, 5010, 5018, 5021,
5023, 5026, 5044, 5060, 5108
- * (Epid-Biom): 5152

- 4993 CYTOTOXIC ANTIBODIES IN RETICULOSSES.
(Fr.) Moulinier, J. (Reg. Ctr. Blood Transfusion, Bordeaux, France), B. Hoerni, M.-C. Merle and C. Mothey. *Bordeaux Med* 5(11):1291-1294, 1972.

Cytotoxic antibodies were detected in sera from 57 of 121 patients with tumors of the reticulo-endothelial system. These patients consisted of 91 with Hodgkin's disease and 30 with reticulum cell sarcomas or lymphosarcomas. Although the presence of cytotoxic antibodies could be accounted for in 31 patients who had either received blood transfusions or been pregnant, the remaining 26 patients had no pertinent history. Cytotoxic antibodies occurred more commonly in men (35 of 68) than in women (20 of 53); no correlation was found between the presence of these antibodies and age. Cytotoxic antibodies were present in about the same percentage of patients with Hodgkin's disease (44 of 91) and those with other reticulososes (13 of 30). However, cytotoxic antibodies were significantly more common among patients with progressive forms (39 of 60) than among patients with remissions (18 of 61). This finding and the failure to elicit pertinent histories from 26 patients with cytotoxic antibodies suggests that these antibodies are associated with reticulososes. Tests for cytotoxic autoantibodies, performed on 36 patients, were positive in 19 (13 with Hodgkin's disease and 6 with other reticulososes), confirming that these patients secrete abnormal immunoglobulins. No correlation was found between the presence of autoantibodies and the WBC or positive BCG tests. The Coomb's test was positive in only one patient with cytotoxic antibodies and a reticulum cell sarcoma. It is suggested that the high incidence of cytotoxic antibodies and autoantibodies in reticulososes results from either proliferation of suppressed clones which are liberated by an immune deficiency or from a defensive reaction to malignant reticular cells which probably have antigens differing from those in the human lymphocyte antigen system. These two hypotheses are not incompatible.

- 4994 *IN VITRO* STUDIES OF LYMPHOCYTES FROM PATIENTS WITH PLASMA CELL MYELOMA. I. STIMULATION BY MITOGENS AND CYTOTOXIC ACTIVITIES. (E.) Mellstedt, H. (Dept. Med., Seraphimer Hosp., Stockholm, Sweden) and G. Holm. *Clin Exp Immunol* 15(3):309-320, 1973.

Highly purified blood lymphocytes from patients with plasma cell myeloma were tested in different *in vitro* systems. The patients were untreated or had received a standardized 4-day treatment with melphalan and prednisolone every sixth week. Lymphocytes from healthy controls were also studied. Stimulation of the lymphocytes was measured by the incorporation of ^{14}C -thymidine into DNA following activation with phytohemagglutinin (PHA), concanavalin A (Con A), and pokeweed mitogen (PWM). Their cytotoxicity was tested against Chang cells (human cell line) and chicken red blood cells in the presence of PHA or heat-inactivated rabbit antibodies to target cell antigens. Lysis was quantitated as the release of radioactivity from target cells labelled with ^{51}Cr -chromate. The antibody-induced cytotoxicity of lymphocytes from the untreated patients was normal or slightly ele-

vated, while that of the treated patients was severely depressed. In addition, the lymphocytes from the treated patients were significantly less stimulated to DNA synthesis by PWM than were the control lymphocytes. PHA-induced cytotoxicity and the stimulation of lymphocytes by Con A or PHA were normal in all groups. These data suggest that the treatment of myeloma patients with melphalan and cortisone selectively impairs lymphocytes which respond to PWM by DNA synthesis and which participate in antibody-mediated cytotoxicity. This impairment may involve the selective removal of such lymphocytes or their functional impairment.

- 4995 STRAIN SPECIFIC CELL-MEDIATED RESPONSE TO POLYOMA-TRANSFORMED FIBROBLASTS IN MICE.
(E.) Molinari, J. A. (Sch. Dental Med., U. Pittsburgh, Pa.), J. L. Ebersole and D. Platt. *Arch Gesamte Virusforsch* 43(1-2):161-164, 1973.

NIH Swiss and BALB/c mice were immunized with an established line of polyoma virus-transformed 3T3 mouse fibroblasts (PY-3T3) and were challenged 7 days later to detect positive delayed hypersensitivity reactions. Another group of mice (uninoculated controls) was injected with saline and 7 days later injected in one hind foot pad with 3T3 or PY-3T3 fibroblasts, while a third group (transplantation antigen group) was immunized with normal 3T3 cells and challenged with the same cell line in the same manner as the uninoculated controls. Upon challenge, 27 of the 37 NIH Swiss mice showed a delayed immune reaction, as opposed to only 1 of the 42 BALB/c mice. The positive response of the NIH animals was significantly different from that shown by the NIH controls and BALB/c experimental and control animals; the responses of the BALB/c controls did not differ from that of the experimental animals. There were negligible differences between the tumor cell and saline-treated foot pads of the unimmunized controls and there was no significant difference in the response of the uninoculated control groups and the transplantation antigen control groups toward the normal 3T3 cells. Thus, the cell-mediated immune reaction observed in the NIH animals was not directed against transplantation antigens.

- 4996 THE PROTECTIVE EFFECT OF ANTISERA AGAINST LEUKAEMIA *IN VIVO*-A REAPPRAISAL. (E.) Hersey, P. (Radcliffe Infirmary, Oxford, England). *Br J Cancer* 28(1):11-18, 1973.

The ability of antibody to cooperate with certain nonimmune lymphoid cells in the rejection of tumors was studied both *in vitro* and *in vivo*. Antiserum raised in rabbits against a PVGc hooded rat leukemia can be rendered tumor specific by *in vivo* absorption in normal PVGc rats and this antiserum can be shown to have lymphocyte dependent antibody (LDA) activity against the tumor cell *in vitro*. Complement lytic activity of the antiserum is weak or absent against the tumor. Passive administration of the antiserum to rats protects the animals against the development of leukemia. Maximum protection occurs if the anti-

serum is given before the tumor inoculum and is less effective given after the tumor inoculum. No protective effect can be demonstrated with tumor specific allo-antisera or syngeneic antisera against the tumor and no *in vitro* LDA activity can be detected against the tumor *in vitro*. Good correlation exists between LDA activity in the sera of the recipients and the *in vivo* protective effect. Differences in the kinetics of cell killing by antiserum and chemotherapeutic agents are discussed. Present evidence suggests that antiserum therapy will be effective only when the tumor cell load is small. The main place of antiserum in leukemia therapy may thus be after induction of remission by chemotherapeutic agents, with the antiserum possibly resulting in a 2-3 log₁₀ kill of remaining chemotherapy-resistant leukemic cells.

- 4997 LYMPHOCYTE STIMULATION BY AUTOCHTHONOUS HUMAN SOLID TUMOURS. (E.) Stjernswärd, J. (Dept. Tumor Biol., Karolinska Inst., Stockholm, Sweden), F. Vanky and E. Klein. *Br J Cancer* 28(1):72-76, 1973.

The validity of lymphocyte stimulation by fresh biopsy cells from autochthonous human tumors as a test reflecting tumor-associated immunity is analyzed. Various parameters such as immunity, serum-mediated blocking, and tumor specific nonresponsiveness can be measured. However, most cancer patients are exposed to treatments that may change both quantitatively and qualitatively not only the cells used but the whole relevance of *in vitro* findings when extrapolated to the *in vivo* situation. Comparison of the stimulability of circulating blood lymphocytes by autochthonous breast tumor cells before and after postoperative radiation indicated impairment of the remaining population after irradiation. This presumably reflected sublethal irradiation damage in the remaining circulating cells. It is suggested that as few changes as possible be introduced in *in vitro* tests of tumor specific-immunity.

- 4998 CELL-MEDIATED IMMUNE RESPONSES *IN VITRO*. V. A COMPARATIVE STUDY OF *IN VITRO* IMMUNOGENICITY OF SPLENIC LYMPHOCYTES, NEOPLASTIC LYMPHOID CELLS AND FIBROBLASTS. (E.) Wagner, H. (Walter and Eliza Inst. Med. Res., Melbourne, Australia) and C. Wyss. *Eur J Immunol* 3(9):549-555, 1973.

The immunogenicity of mouse lymphocytes, neoplastic lymphoid cells, or fibroblasts was studied in a cytotoxic allograft system in which CBA cortisone-resistant thymocytes were cultured together with mitomycin C-treated stimulator cells of BALB/c origin. In addition, the capacity of mouse spleen cells to stimulate in mixed lymphocyte culture (MLR) was compared with their capacity to stimulate in cytotoxic allograft responses. Spleen cells of neonatal mice showed reduced capacity to stimulate in MLR compared with those of adult mice. However, their capacity to induce cytotoxic allograft responses was as good as that of adult mice. Lymphocytes enriched for B or T cells were equally immuno-

genic *in vitro*, as were myeloma or thymoma lymphoid cells. In contrast, fibroblasts elicited poor cytotoxic allograft responses. The present data do not allow conclusions as to whether there is a qualitative difference between the immunogenicity of lymphoid cells and fibroblasts, or whether a mere quantitative H-2 antigen difference is expressed on the cell surface of the different cell types.

- 4999 A STUDY OF METHODS FOR PRODUCING CELL-FREE TUMOUR ANTIGEN FROM BP8 MOUSE ASCITES TUMOUR. (E.) Davies, E. G. (Dept. Path., U. Cambridge, England) and D. B. Cater. *Br J Exp Pathol* 54(5):583-589, 1973.

One million lymph node cells (LNC) from C57 Bl mice immunized with BP8 cells plus Freund's complete adjuvant (FCA) protected C3H mice against fatal challenge with BP8 injected i.p. 20 hours later. The LNC of C57 Bl mice immunized with the supernatant from BP8 cells treated with lysolecithin (1.5 µg/10⁶ cells) gave equally good protection. BP8 cells after treatment with lysolecithin equalled untreated BP8 cells in antigenic efficiency. Cell-free preparations made from BP8 cells with 3 molar KCl were of doubtful antigenic efficiency. The LNC from C57 Bl mice immunized with C3H liver and spleen tissue plus FCA gave no protection and LNC from non-immunized C57 Bl mice did not protect C3H mice against BP8 tumour. Centrifugation of the supernatants at 105,000 g indicated that the antigenic properties of the lysolecithin supernatant might be due to subcellular particles which were not found in the 3 molar KCl preparations.

- 5000 CROSSED REACTION OF HUMAN BREAST CANCER AND FIBROCYSTIC MASTOPATHY WITH MURINE MAMMARY CARCINOMAS: LOCATION OF THE ANTIGEN IN A-PARTICLES OF MAMMARY TUMOR VIRUS. (E.) Mueller, M. (Carl Gustav Carus Med. Acad., Dresden, Germany), C. Kemmer, S. Zotter, H. Grossmann and B. Michael. *Arch Geschwulstforsch* 41(4):100-106, 1973.

The indirect immunoferritin technique was used to determine whether antibodies found in women with breast cancer and fibrocystic mastopathy react with intracellular clusters of A-particles which are immature precursors of the B-particles of mouse mammary tumor virus. Mouse tumor MT4814, obtained by passage in syngeneic CBA/Bl mice was fixed in neutral 4% formalin by shaking for 10 min. Cells obtained in this way were washed three times in phosphate-buffered physiological saline and incubated for 3 hr at room temperature with 0.1 ml of C89 serum from a patient with fibrocystic mastopathy, blood donor serum, or phosphate-buffered physiological saline. After washing three times with physiological saline, cells were incubated for 2 hr at room temperature with 0.1 ml ferritin-serum. Examination under the electron microscope showed that ferritin granules were deposited only in tumor cells incubated with C89 serum. These granules were present in clusters of A-particles or surrounded individual A-particles or groups of them. Ferritin

deposits on the cytoplasmic membrane are also considered specific since they were not present in controls. No ferritin deposits occurred on B-particles. These results, which agree with those obtained with immunofluorescence techniques, provide evidence for the existence of a human breast cancer virus which is immunologically related to mouse mammary tumor virus.

- 5001 STUDY OF THE THYMIC-DERIVED OR -INDEPENDENT NATURE OF MOUSE SPLEEN CELLS INDUCED TO PROLIFERATE IN CULTURE BY VARIOUS MITOGENS AND ANTIGENS. (E.) Piguet, P.-F. (Dept. Pathol., U. Geneva, Switzerland) and P. Vassalli. *Eur J Immunol* 3(8):477-483, 1973.

The nature of mouse lymphoid cells induced to proliferate *in vitro* by a number of stimulating agents has been explored by 1) karyotypic analysis of the mitoses observed in stimulated spleen cell cultures (arrested on sequential days) of chimaeras bearing chromosomally marked T cells; 2) comparison between the response in DNA synthesis of total spleen cells and of spleen cells depleted of thymus-dependent lymphocytes by treatment with anti-MSLA (anti-mouse-specific lymphocyte antigen) + C, and 3) study of the response of cortisone-resistant thymocytes (CRT) to some of these agents. Pokeweed mitogen stimulated both B and T cells equally and simultaneously. Rabbit anti-mouse Ig induced moderate proliferation of B cells exclusively. The response to lipopolysaccharide was entirely B cell-dependent, but evidence was found of some T cell proliferation following or accompanying the stimulation of B cells. A similar observation was made in the case of "primary" or "secondary" *in vitro* response to bacteriophage T4. Good stimulation with sheep red blood cell stromas or mitogen-treated xenogenic spleen cells were observed only in cultures from primed animals, and consisted of a mixed B and T cell proliferation with a majority of B mitoses, even at the earlier stages of the response. That this "secondary" response was not totally T cell-dependent was suggested by the observation of a stimulatory effect of these antigens on T-depleted spleen cell cultures which were found in parallel experiments to be totally unresponsive to PHA. Finally, using chimeric mice treated *in vivo* with vinblastine prior to establishment of the spleen cell cultures, evidence was obtained that part of the B cells stimulated to divide *in vitro* by several of these mitogens belong to a rapidly dividing cell population *in vivo*.

- 5002 NUDE MICE--A MODEL SYSTEM FOR STUDYING THE CELLULAR BASIS OF THE HUMORAL IMMUNE RESPONSE. (E.) Croy, B. A. (Inst. Med. Sci. Dept. Med., U. Toronto, Canada) and D. Osoba. *Cell Immunol* 9(2):306-318, 1973.

Mice homozygous for the *nu* gene fail to develop a thymus. In comparison to spleen cells from *+/nu* mice spleen cells from *nu/nu* mice have a deficient 19S plaque-forming cell (PFC) response to sheep red blood cells (SRBC) when tested in culture or

in vivo. This deficiency is due to a lack of "helper" T cells in *nu/nu* spleen; A cells and B cells appear to be normal. The capacity of *nu/nu* spleen cells to produce a PFC response in culture can be corrected by the addition of T cells obtained from either the thymuses or the spleens of *+/nu* mice. In contrast to "helper" T cells obtained from the spleen, "helper" T cells obtained from the thymus appear to require the capacity for proliferation during the response to SRBC.

- 5003 THE POSSIBLE ROLE OF THE THYMUS IN CFU PROLIFERATION AND DIFFERENTIATION. (E.) Frindel, E. (Inst. Gustave Roussy, Villejuif, France) and H. Croizat. *Biomedicine* 19(9):392-394, 1973.

Thymectomy and sham thymectomy were performed on C₃H female mice at 8 wk of age. Stem cell proliferation was assessed by the tritiated thymidine suicide method. After sham thymectomy the C₃H mice had about 20% of their bone marrow stem cells in DNA synthesis under standard holoxenic conditions. After thymectomy, the proportion of CFU in DNA synthesis dropped to practically zero as early as 7 days after operation and remained there for 8 months. Salmonella 0 antigen challenge in sham thymectomized mice increased the proportion of stem cells in DNA synthesis from 20-33%. No response to antigenic challenge occurred in thymectomized mice. When challenged with bleeding, the thymectomized mice had an increase in the proportion of these cells to 28%, and in the sham operated mice bleeding triggered CFU's into S and the proportion of these cells increased to 38%. These results suggest that adult thymectomy suppresses CFU response to antigenic stimulation. Apparently the mechanism for CFU stimulation by bleeding differs from the mechanism by antigen stimulation, as the latter is without effect in thymectomized mice.

- 5004 CORRELATION AMONG HOST IMMUNOCOMPETENCE AND TUMOR STAGE, TUMOR GRADE AND VASCULAR PERMEATION IN TRANSITIONAL CARCINOMA. (E.) Catalona, W. J. (Johns Hopkins Hosp., Baltimore, Md.) and P. B. Chretien. *J Urol* 110(5):526-528, 1973.

Dinitrochlorobenzene (DNCB) was used in contact sensitization in 25 patients with transitional carcinoma of the urinary tract and in 8 tumor-free patients with histories of transitional cell tumor, in order to evaluate cell-mediated immune response. A 56% incidence of impaired reactivity was noted in tumor-bearing patients, while tumor-free patients had normal immune reactivity. A highly significant correlation was demonstrated between the standard parameters used in assessing tumor aggressiveness (i.e., tumor stage, tumor grade and vascular or lymphatic permeation) and impaired host cell-mediated immuno-competence. The data do not reveal whether the lack of the cell mediated immunologic responsiveness observed in the tumor-bearing patients resulted from or was the cause of more

advanced malignancy. However, the results do show that evaluation of host immuno-competence offers a useful means of gauging the biologic potential of a tumor.

- 5005 FOREIGN ANTIGENICITY IN TISSUES OF MICE INFECTED WITH A LYMPHOMAGENIC VIRUS. I. ANTIGENICITY OF SPLEEN CELLS. (E.) Salaman, M. H. (Royal Coll. Surg. England, London), J. L. Turk and N. Wedderburn. *Transplantation* 16(6):583-590, 1973.

Normal BALB/c mice were infected with the lymphomagenic virus, urethane lymphoma virus (ULV). Their spleens acquired a new antigenicity, as evidenced by wt increase of popliteal lymph nodes (PLNs) of syngeneic normal recipients 7 days or more after the infected spleen cells were injected into their hind footpads. Antigenicity was noted during the 1st wk after infection of the donors. Simultaneously, infected mice lose the ability to react by PLN enlargement to footpad injection of spleen cells from donors infected with the virus 2 wk or more previously. X-irradiation *in vitro* reduced but did not abolish the antigenicity of infected spleen cell suspensions. Virus preparations freed of cell debris are not antigenic. Nonreactivity of infected mice to footpad inoculation of infected spleen cells is not caused by a nonspecific immune depression by the virus; for infected mice show no significant depression of reactivity, in the PLN test, to allogeneic spleen cells differing at a non-H-2 locus. Moloney lymphomagenic virus and a lymphomagenic virus isolated from a spontaneous lymphoma in an old BALB/c mouse both induced spleen cell antigenicity qualitatively similar to that obtained for ULV.

- 5006 BLASTOID LYMPHOCYTE TRANSFORMATION ASSAY IN MALIGNANT TUMOURS OF LYMPHOPOIETIC TISSUE: IN HODGKIN'S DISEASE, RETICULOSARCOMA, LYMPHOSARCOMA AND CHRONIC LYMPHATIC LEUKAEMIA. RELATIONSHIP TO THE CLINICAL STATUS AND TO THE DELAYED TYPE OF HYPERSENSITIVITY. (E.) Libansky, J. (Inst. Hematology Blood Transfusion, Prague, Czechoslovakia). *Folia Haematol (Leipz)* 100(1/2):51-56, 1973.

The relationship between the clinical status of patients affected with malignant diseases of the lymphatic system (Hodgkin's disease, reticulo- and lymphosarcoma and chronic lymphatic leukemia) as well as between PHA-induced transformed lymphocytes and the ability to respond to skin tests with tuberculin and streptokinase by a delayed hypersensitivity were examined. In patients with Hodgkin's disease the response to PHA was independent of absolute lymphocyte count in the blood. In chronic lymphatic leukemia patients the values of lymphocyte transformation were very low. The range of values was not as wide as in Hodgkin's disease or in reticulosarcoma. Lymphocyte response to PHA was studied in 31 patients, 24 of whom were in the active disease stage and 7 in remission. The mean transformation index in the group of symptomatic patients was significantly lower than

that in healthy controls. The lack of correlation with absolute lymphocyte count in the blood was noted nor was there a correlation with therapy. Only a partial correlation was found between the response to PHA and the results of tuberculin and streptokinase skin tests. Further examination in the same patients at different stages of their diseases are therefore necessary for a definite answer to be given.

- 5007 SUPPRESSED DEVELOPMENT OF CYTOTOXIC LYMPHOID CELLS IN TUMOR-IMMUNIZED MICE. (E.) Martin, W. J. (Natl. Cancer Inst., Bethesda, Md.), J. Wunderlich and J. MacDonald. *Isr J Med Sci* 9(3):324-331, 1973.

The *in vivo* and *in vitro* immune responsiveness of C57B1/6 mice to a syngeneic lymphoid leukemia, EL-4, was analyzed. Immunization by two i.p. injections of irradiated EL-4 (5×10^5 and 5×10^7) cells evoked no antitumor cytotoxic lymphoid cell activity. Immunization with EL-4 cells that had undergone reaction with Concanavalin A (Con A), followed by injection of uncoated EL-4 cells, led to high levels of cytotoxic lymphoid cell activity. Spleen cell populations from normal mice or mice previously injected with uncoated or Con-A coated EL-4 cells responded *in vitro* to EL-4 cells with development of cytotoxic lymphoid cells. Furthermore, spleen cells from preimmunized mice sometimes spontaneously developed tumor-specific cytotoxic lymphoid cell activity following *in vitro* culture. These findings suggest that in tumor-immunized mice the development of cytotoxic lymphoid cells is suppressed *in vivo*; this suppressive mechanism is less evident in *in vitro* systems. A practical approach to immunotherapy may be to bypass the *in vivo* blocking mechanism by using adoptively transferred cytotoxic lymphoid cells. It is suggested that these cells could be obtained by *in vitro* sensitization via lymphoid cells from tumor-immunized hosts.

- 5008 A MATHEMATICAL MODEL FOR THE QUANTITATION OF TUMOR AND ANTIBODY-FORMING CELL POPULATIONS WITHIN AN ANIMAL'S BODY. (E.) Hiramoto, R. N. (U. Alabama Med. Ctr., Birmingham), V. K. Ghanta, J. R. McGhee and N. M. Hamlin. *J Immunol* 111(5):1546-1553, 1973.

The application of a mathematical model described by Hege and Cole was tested by a murine myeloma model. The plasmacytoma MOPC 104E secretes an IgM antibody to bacterial dextran B-1355. By using this system the various parameters required in the equation could be quantitated. The model relates the number of tumor cells in the population to antibody produced under conditions of logarithmic growth. Experimental values of antibody measured and estimation of starting population (A_n) of tumor cells showed that 2.24×10^6 cells grew (days 2-14) when 3×10^6 cells were injected in BALB/c mice. This indicates that approximately 75% of the cells grew when 3.0×10^6 tumor cells were given i.p. into each mouse. The experimentally determined antibody

levels were in agreement with the computer calculated values.

- 5009 QUANTITATIVE STUDY OF ERYTHROCYTE ANTIGENS I AND i IN PATHOLOGY. (Fr.) Rochant, H. (Henri Mondor U. Hosp. Ctr., Creteil, France), H. Tonthat, N. M. Man, J. Lefaou, A. Henri and B. Dreyfus. *Nouv Rev Fr Hematol* 13(3):307-318, 1973.

Erythrocyte antigen I determinations were made on 376 subjects and antigen i determinations on 515 subjects. Most of the patients studied had malignant or premalignant blood diseases; controls consisted of elderly subjects, normal adults, and newborns. Antigen I was significantly increased in acute leukemia and, to a somewhat lesser extent, in chronic myeloproliferative syndromes (chronic myeloid leukemia, primary myelofibrosis, polycythemia vera). Levels of antigen I were increased in megaloblastic anemia resulting from vitamin B₁₂ or folate deficiency but were normal in refractory anemia, iron-deficiency hypochromic anemia, and 10 patients with cancer. No significant differences were found in antigen I before and after chemotherapy and/or radiotherapy. Antigen I levels were not correlated with age. Decreased antigen I levels only occurred in 7 of 376 cases. Erythrocyte antigen i was increased in 14 of 40 patients with acute leukemia, in 14 of 34 with chronic myeloproliferative disease, in 11 of 14 with bone marrow aplasia, in 17 of 25 with refractory anemia, and in all 106 of the newborns examined. This finding suggests that fetal synthesis of antigen i is resumed in malignant or premalignant blood diseases. Since the bone marrow is not directly affected by cellular proliferation, increases in antigen I may be caused by environmental factors. Because the I system is a precursor of A, B and H substances, it is also possible that antigen I is unmasked by a decrease in the synthesis of ABO blood-group substances. A positive correlation was found between increases in antigen I and increases in antigen i.

- 5010 STUDIES ON INTRACELLULAR AND MEMBRANE ANTIGENS INDUCED BY MAREK'S DISEASE VIRUS. (E.) Nazerian, K. (U.S. Dept. Agriculture, Regional Poultry Res. Lab., East Lansing, Mich.). *J Gen Virol* 21(1):193-195, 1973.

Prolonged passage of Marek's disease virus (MDV) in cell culture resulted in attenuation of the virus and inability of the virus to produce the membrane antigen in chick kidney cultures. However, the apathogenic herpes virus of turkeys produced a serologically similar membrane antigen. Cell culture passage did not cause any change in production of intracellular antigens. The absence of membrane antigen in the presence of intracellular antigens in cells infected with attenuated strains of MDV and the difference in antibody titers to these antigens indicate that they are probably two different antigens. The membrane antigen may be related to the 'A' antigen detected in immunoprecipitin test, since both are lost as the virus is serially passaged in cell culture and becomes attenuated.

- 5011 IMMUNOCHEMICAL STUDIES OF THE INTRAMOLECULAR HETEROGENEITY OF THE CARCINOEMBRYONIC ANTIGEN (CEA) OF THE HUMAN DIGESTIVE SYSTEM. (E.) Gold, J. M. (Montreal Gen. Hosp., Canada), C. Banjo, S. O. Freedman and P. Gold. *J Immunol* 111(6):1872-1879, 1973.

Previous studies have shown that, in addition to the tumor-specific site on the carcinoembryonic antigen (CEA), this molecule contains a blood group A-like grouping. To study the A-like site on the CEA molecule, a radioimmunoassay in which anti-A antibodies were coupled to either Sepharose or Sephadex beads was devised. The monosaccharide, *N*-acetyl-D-galactosamine was capable of inhibiting the interaction between ¹²⁵I-CEA and a preparation of anti-A antibodies. In addition, a glycopeptide (GP-1), containing the tumor-specific antigenic site of the CEA, which was obtained by the enzymatic degradation of the CEA molecule was capable of binding to anti-A antibodies. The ratio, by weight, of GP-1 to *N*-acetyl-D-galactosamine required to achieve equivalent binding was 10⁻³ to 10⁻⁴ to 1. GP-1 has a molecular weight of about 4000 daltons, and although it contains *N*-acetyl-D-glucosamine, D-mannose, D-galactose, and L-fucose, the glycopeptide is apparently devoid of *N*-acetyl-D-galactosamine. Thus, at least some of the carbohydrate chains of the CEA molecule may well possess tumor-specific and A-like antigenic specificities.

- 5012 IMMUNOLOGIC TESTS FOR THE DETECTION OF GASTROINTESTINAL CANCERS: STATUS REPORT ON CARCINOEMBRYONIC ANTIGEN (CEA) AND ALPHA-FETOPROTEIN (AFP). (E.) Groover, J. R. (U. Miami Sch. Med., Florida) and A. I. Rogers. *South Med J* 66(11):1218-1221, 1973.

Advances have been and continue to be made in cancer detection employing a variety of simple and sophisticated serum assays for tumor antigens. Whether or not these antigens are tumor-specific may not be as important a consideration as that they are usually tumor-associated. Certain clinical circumstances such as advanced alcoholic liver disease, alcoholic pancreatitis, and uremia, in the case of carcinoembryonic antigen, may result in seropositivity in the absence of tumor. The clinician should be aware of these circumstances so that he can assign accurate significance to seropositivity. The CEA assay appears to be of primary use in the assessment of the adequacy of surgical resection, evaluation of extent of metastases, and, in certain situations, screening for the presence of malignancies. Alpha-fetoprotein seropositivity has been described in patients with prostatic and gastric carcinomas metastatic to the liver, in neonatal hepatitis, and in adults with overwhelming acute viral hepatitis. Overall incidence of alpha-fetoprotein seropositive reactions in humans with hepatomas has varied from 40 to 80%. In addition to usefulness of such assays in cancer detection and assessment of therapeutic responsiveness, serodiagnosis has paved the way for enhanced understanding of tumor immunogenesis and possible immunotherapy.

- 5013 SPECIFIC CYTOSTATIC EFFECT OF LYMPH NODE CELLS FROM NORMAL AND T CELL-DEFICIENT MICE ON SYNGENEIC TUMOR TARGET CELLS *IN VITRO* AND ITS SPECIFIC ABROGATION BY BODY FLUIDS FROM SYNGENEIC TUMOR-BEARING MICE. (E.) Chia, E. (London Hosp. Med. Coll., England) and H. Festenstein. *Eur J Immunol* 3(8):483-487, 1973.

Lymphoid cells from tumor-bearing animals (inbred BALB/c and DBA/2 mice, 8-12 wk old) were shown to be cytostatic for syngeneic tumor target cells *in vitro* using a post labeling radioactive assay. The specific decrease of removal of this cytostatic activity was possible using a "blocking" serum or ascitic fluid from syngeneic tumor-bearing animals. Lymphoid cells from chronically irradiated, thymectomized mice, which are shown to be extensively deprived of T cells by reduced phytohemagglutinin responsiveness and anti- ϕ cytotoxicity tests, were found to cause cytostasis of target tumor cells to a greater extent than non-deprived lymphoid cells. The length of time required (72 hr) to demonstrate maximum cytostasis of target cells may indicate an *in vitro* secondary response of the lymphoid cells to tumor antigens, after the primary *in vivo* sensitization.

- 5014 STUDIES ON HETEROPHILE ANTIGEN IN LYMPHOMA AND LEUKEMIA SPLEENS BY MEANS OF INFECTIOUS MONONUCLEOSIS SERA. (E.) Milgrom, F. (Sch. Med., St. U. New York at Buffalo), K. Kano and A. Fjelde. *Int Arch Allergy* 45(4):631-637, 1973.

Spleen specimens from patients suffering from lymphomas and leukemias were studied for the presence of heterophile antigen by means of absorption of infectious mononucleosis sera. In the lymphoma group, 11 of 16 spleens tested diminished significantly the titer of lysins for bovine red blood cells in infectious mononucleosis sera. The absorbing effect of these spleen specimens correlated well with the replacement of the normal organ structure by malignant cells. None of five spleens from leukemia patients had a significant absorbing effect on infectious mononucleosis sera. The results of this study seem to indicate that malignant transformation of human lymphocytes may be accompanied by acquisition of the heterophile antigen. It cannot be explained at present why patients with lymphoma fail to form heterophile antibodies. It is possible that the mechanism which prevents formation of heterophile antibodies by patients suffering from lymphoma (and possibly also from leukemia) may be of basic importance for the malignant character of these diseases.

- 5015 AN IMMUNOLOGIC COMPARISON BETWEEN SEROUS CYSTADENOCARCINOMA OF THE OVARY AND OTHER HUMAN GYNECOLOGIC TUMORS. (E.) Bhattacharya, M. (Roswell Park Memorial Inst., Buffalo, N.Y.) and J. J. Barlow. *Am J Obstet Gynecol* 117(6):849-853, 1973.

Absorbed rabbit antiserum with specific reactivity against human serous cystadenocarcinoma of the

ovary was reacted with tissue extracts of various gynecologic cancers and malignant ovarian cyst fluids by the direct Ouchterlony double-diffusion and precipitin-inhibition techniques. In all specimens tested, serous cystadenocarcinomas and mucinous cystadenocarcinomas of the ovary were immunologically indistinguishable. Each contained at least two tumor-associated antigens. The major precipitin band was shared by and appeared to be specific for serous and mucinous cystadenocarcinomas, whereas a faint precipitin band cross reacted with other tumors of the reproductive tract. An identical antigenic pattern was demonstrated for the cyst fluids of serous and mucinous cystadenocarcinomas. On the basis of these findings, the antigen(s) represented by the major precipitin band may be considered a common ovarian cystadenocarcinoma-associated one. It is of interest that this antigen could not be demonstrated in endometroid ovarian cancers because this tumor is also thought to arise in common with serous and mucinous ovarian tumors from the germinal epithelium.

- 5016 INTRODUCTION OF CELLULAR IMMUNITY TO A CHEMICALLY ALTERED TUMOR ANTIGEN. (E.) Choa, H.-F. (Washington U. Sch. Med., St. Louis, Mo.), S. C. Peiper, R. D. Aach and C. W. Parker. *J Immunol* 111(6):1800-1804, 1973.

Hartley strain guinea pigs were immunized with unaltered human carcinoembryonic antigen (CEA) and CEA acetoacetylated with acetoacetic anhydride (AA-CEA). Two weeks later, all animals were injected intradermally with 5 μ g of purified CEA and observed for skin reactions. Immunization with CEA resulted in both delayed hypersensitivity and humoral immune responses to CEA; immunization with AA-CEA resulted in evidence of delayed hypersensitivity with only a minimal humoral antibody response to CEA. Radioimmunoassay studies indicated that much more anti-CEA antibody was present in the serum of the animals immunized with CEA than in the AA-CEA-immunized animals. Acetoacetylation may be useful in further defining the role of humoral- and cell-mediated immunity in tumor growth in experimental animals.

- 5017 IMMUNOCHEMICAL STUDIES ON CARCINOEMBRYONIC ANTIGEN (1) QUANTITATIVE DETERMINATION IN CARCINOMATOUS AND NONCARCINOMATOUS TISSUES. (E.) Kawaharada, M. (Sapporo Med. Coll., Japan), A. Araki, A. Yachi and T. Wada. *Jap J Clin Oncol* 6(3):31-40, 1973.

Anti-carcinoembryonic (CEA) serum was prepared by immunizing rabbits with a crude CEA fraction obtained from perchloric acid-soluble extracts of adenocarcinoma of the stomach. After absorption, the antiserum proved to be monospecific for CEA. With Mancini's technique, CEA concentrations in perchloric acid extracts of carcinomatous and noncarcinomatous tissues were quantitatively determined and expressed in terms of unit/ml, equivalent to mg/ml protein concentration of standard CEA solution. Finally, ratios of CEA units/mg protein (CEA/P) of tissue extract were calculated. The CEA/P ratios tended

to be higher in extracts from colonic (10), rectal (5) and pancreatic (2) carcinomas than in those from gastric carcinoma (20), whereas they were lower in those from primary hepatoma (3) and lung cancer (3 patients). In the same patients with cancer, the ratios for carcinoma tissues were remarkably higher than those for the noncarcinomatous counterparts. However, the ratios were not related to the histological types of cancers. Relatively high ratios were also observed in diseased intestinal mucosa of colonic polyposis and Crohn's disease, as well as in parts of gastric mucosa with a marked intestinal metaplasia obtained from a cancerous stomach. More studies will be required to evaluate the significance of occurrence in precancerous changes in tissues.

- 5018 ALLOGENEIC BONE MARROW CHIMERISM IN GERM-FREE MICE. 1. PREVENTION OF SPONTANEOUS LEUKEMIA IN AKR MICE. (E.) Pollard, M. (Lobund Lab., U. Notre Dame, Ind.) and R. L. Truitt. *Proc Soc Exp Biol Med* 144(2):659-665, 1973.

At 11 weeks of age, two groups of germfree (GF) AKR mice were X-irradiated. Twenty-four hours later, each mouse was injected i.v. with 10,000,000 pooled viable bone marrow cells from the femurs of mature GF DBA/2 mice. Control AKR groups were: untreated; given X-ray treatment only; irradiated and injected with AKR bone marrow cells; or not irradiated and given bone marrow cells from conventional DBA/2 mice. All mice subjected only to the X-irradiation procedure died within 13 days, while mice irradiated and inoculated within 24 hours with viable syngeneic bone marrow cells survived the acute radiation disease syndrome for varying periods, after which they developed spontaneous leukemia. The conventional AKR animals inoculated with bone marrow cells from conventional DBA/2 mice appeared healthy for several days then began developing symptoms of graft-versus-host (GVH) reaction. In contrast, the same type of incompatible bone marrow chimerism was relatively innocuous in the GF mice; although they frequently appeared dyspneic and kyphotic because of accumulated clear fluid in the thorax and some showed evidence of mild GVH disease, none developed spontaneous leukemia up to age 15 months (beyond the age incidence of leukemia in AKR mice), when the experiment was terminated. Thus leukemia was prevented in the GF AKR mice with allogenic chimerism, suggesting that adoptive immunotherapy, under carefully controlled conditions, may prove useful as an adjunct to conventional antileukemia therapy.

- 5019 THE ASSOCIATION OF AUTOIMMUNE THROMBOCYTOPENIA AND HODGKIN'S DISEASE. (E.) Khilanani, P. (Sch. Med., Wayne State U., Detroit, Mich.) and M. Al-Sarraf. *Oncology* 28(3):238-245, 1973.

A 24-year-old Caucasian male with a palpable mass at the right submandibular area presented a 6-year history of a bleeding tendency characterized by frequent episodes of epistaxis, bruising, and purpura. A biopsy of the submandibular mass revealed Hodgkin's

disease, stage III-A, of mixed cellular type, and the bleeding tendency was subsequently diagnosed as autoimmune thrombocytopenia. The thrombocytopenia initially responded to steroid therapy followed by splenectomy; it is now responding to cytoxan therapy. Treatment of the Hodgkin's disease with radiation had no effect on the course of the autoimmune thrombocytopenia, even though the former is in complete remission. Serum analysis was positive for platelet factor-3 antiplatelet autoantibody, and no isoantiplatelet antibody was found as a result of the complement fixation test. The development of Hodgkin's disease in this patient after a 6-year history of autoimmune thrombocytopenia may be more than coincidental.

- 5020 IN VITRO STUDIES OF LYMPHOCYTES FROM PATIENTS WITH PLASMA CELL MYELOMA. II. CHARACTERIZATION BY CELL SURFACE MARKERS. (E.) Mellstedt, H. (Seraphimer Hosp., Stockholm, Sweden), M. Jondal and G. Holm. *Clin Exp Immunol* 15(3):321-330, 1973.

Highly purified blood lymphocytes from treated and untreated patients with plasma cell myeloma were tested for the presence of thymus-derived lymphocytes and non-thymus-derived (EAC) lymphocytes carrying the C3 receptor surface marker. The treated patients were tested 5 wk after a standardized 4-day treatment with melphalan and prednisolone. The percentage of EAC lymphocytes in the peripheral blood of the untreated patients was 40 to 65%, while the percentage in the blood of the treated patients was less than 20%. The percentage in a nonmyeloma control group was 20 to 40%. The lymphocyte mediated lysis of target cells (Chang cell line or chicken red blood cells) treated with rabbit antibody was positively correlated with the percentage of EAC lymphocytes. DNA synthesis induced in lymphocytes by pokeweed mitogen (PWM) showed a biphasic dose-response curve which may indicate the activation of two populations of lymphocytes; a positive correlation was found between the percentage of EAC lymphocytes and the DNA synthesis induced by 0.1 µg/ml PWM. Thus, antibody induced cytotoxicity appears to require cells in the non-thymus derived lymphocyte population, and PWM appears to be partly a mitogen for human bone marrow derived lymphocytes.

- 5021 QUANTITATION OF MOUSE MAMMARY TUMOR VIRUS (MTV) VIRIONS BY RADIOIMMUNOASSAY. (E.) Cardiff, R. D. (U. California, Sch. Med., Davis). *J Immunol* 111(6):1722-1729, 1973.

The radioimmune precipitation (RIP) technique has been applied to the quantitation of the mouse mammary tumor virus (MTV); the MTV is externally labelled with radioiodine via the lactoperoxidase procedure. ¹²⁵I-MTV derived from the milk or mammary tumor cells of BALB/cFC3H (MTV-infected) mice was co-precipitated, specifically in the presence of antibodies against MTV, by goat antibodies to rabbit IgG. The RIP of ¹²⁵I-MTV was inhibited with unlabelled MTV, but it was not inhibited by cell-free extracts of host cell, milk, any of the murine C-

type particles used, non-immune sera, or antibodies against BALB/c spleen cells. The assay reproducibility detected as little as 5 ng of intact virus. The serum inhibition of the RIP can be used to survey mice, rats, humans, and others for the presence or absence of anti-MTV antibodies, and the RIP system shows great potential for studying the antigenic relationships between closely related MTV substrains.

- 5022 FAMILIAL GASTRIC CANCER AND IMMUNOLOGIC ABNORMALITIES. (E.) Creagan, E. T. (Nat'l. Cancer Inst., Bethesda, Md.) and J. F. Fraumeni, Jr. *Cancer* 32(6):1325-1331, 1973.

Stomach cancer developed in 12 members of an inbred family over four generations. The family resided in a rural county in southwestern Virginia where the reported mortality from this cancer has been significantly higher than in the surrounding region, but no specific environmental influence could be identified. A battery of laboratory studies applied to 16 family members, including one recently diagnosed with stomach cancer, revealed a high frequency of cases with antibody to gastric parietal cells and cell-mediated immune defects, as manifested by impaired lymphocyte transformation *in vitro*, skin test anergy, and lymphocytopenia. Although only one member of the family was reported with pernicious anemia, the presence in several family members of parietal cell antibodies and macrocytosis suggests a subclinical process related to pernicious anemia, perhaps a genetically-mediated autoimmune gastritis predisposing to stomach cancer. It is also possible that the inherited susceptibility to stomach cancer in this family is based on a genetically determined defect in thymus-dependent (T) lymphocytes, which mediate cellular immunity.

- 5023 RESISTANCE OF MICE TO INFECTION WITH FRIEND DISEASE VIRUS AFTER SUBCUTANEOUS INJECTION OF FRIEND VIRUS AND FRIEND SPLEEN CELLS. (E.) Larson, C. L. (Dept. Microbiol., U. Montana, Missoula), R. N. Ushijima, S. K. Kasuga, R. E. Baker and M. B. Baker. *Infect Immun* 8(5):708-714, 1973.

Swiss mice injected s.c. with suspensions of spleen cells or an extract of spleens from mice infected with Friend virus develop resistance to subsequent i.v. inoculation of Friend virus. A single injection of either Friend virus or Friend cells induces resistance. Immunized mice display resistance when challenged six months after immunization and survive for at least 20 wk after infection. Neutralization tests indicate that serum, but not lymphoid cells of resistant animals, can neutralize Friend virus. *In vitro* neutralization tests indicate that residence of virus within the peritoneal cavity of immune mice for 1 hr sharply reduces the infective titer of the virus. Resistance which develops after s.c. injection of Friend cells or Friend virus appears to be related to humoral immunity. Administration of Friend virus by the s.c. route may result in a minimal spread of virus through the circulation and in a prompt response in the regional lymph nodes.

- 5024 ANTIBODY CYTOTOXICITY STUDIES IN OVARIAN AND CERVICAL MALIGNANCIES. (E.)

Disaia, P. J. (Los Angeles Cty.-U. Southern California Med. Ctr.), R. H. Nalick and D. E. Townsend. *Obstet Gynecol* 42(5):644-650, 1973.

The cytotoxic effect of serum from patients with progressive squamous cell carcinoma of the cervix and adenocarcinoma of the ovary was evaluated prior to treatment. The cytotoxic ability of six specific rabbit antisera prepared by immunizing rabbits with fresh tissue homogenates of adenocarcinoma of the ovary were also studied and all six demonstrated a definite injurious effect. Twelve of the 20 patients with advanced squamous cell carcinoma of the cervix and five of the eight patients with advanced adenocarcinoma of the ovary also illustrated a definite cytotoxic serum activity. The coexistence of tumor and cytotoxic antibody can probably be explained on the basis of accessibility. The antibody does not readily pass vascular barriers or penetrate intracellular spaces and probably does not reach much of the tumor. In addition, it is conceivable that the amount of tumor may be so massive that antibody is absorbed onto only a portion of the great amount of antigen present, thus leaving little for detection in the circulating blood.

- 5025 AN ATTEMPT TO CHARACTERIZE LYMPHOCYTES OF ACUTE LYMPHOID LEUKAEMIA AS T OR B-CELLS. (E.) Governa, M. (Inst. Industrial Med., U. Genoa, Italy), L. Massimo, C. Rosanda, M. I. Franchini and G. P. Tonda. *Biomedicine* 19(9):384-387, 1973.

In order to classify them as B or T cells, lymphocytes from cases of acute lymphoblastic leukemia were tested for the surface immunoglobulin determinants as a marker of B cells, and for PHA responsiveness as a property of T cells. In the peripheral blood of 15 children with acute lymphoblastic leukemia, cell transformation was marked after PHA stimulation while in most of the cases almost no cells bearing surface immunoglobulins were detected. Therefore, leukemic cells in the peripheral blood of children with this disease are T cells. In bone marrow of these same children no PHA responsiveness was noted and no detectable amount of immunoglobulins were found on the lymphocyte surfaces. Thus lymphocytes in the bone marrow are immature T cells. In the peripheral blood of a 45 yr old woman with this disease, the leukemic cells were B lymphocytes; more than 90% possessed surface immunoglobulins and they failed to respond to PHA.

- 5026 OUTER MEMBRANE OF AVIAN MYELOBLASTOSIS VIRUS. (E.) Ishizaki, R. (Duke U. Med. Ctr., Durham, N.C.), R. B. Luftig and D. P. Bolognesi. *J Virol* 12(6):1579-1588, 1973.

One-month-old guinea pigs were immunized intracerebrally with 1 mg avian myeloblastosis virus (AMV); 4 wk later they received a second inoculation i.p. Their sera produced antiserum which reacted with intact virus particles in complement fixation. The

antigen in question appeared to be located on the surface of the virion and could be distinguished from the type-specific virus envelope and the group-specific internal antigens of chicken leukosis-sarcoma viruses (ChILSV). The material could be isolated by sequential treatments of AMV with bromelain, Tween 20, and freeze-thawing, and could be purified by differential centrifugation. Electron microscopy analysis indicated the presence of a component resembling the outer membrane of the particle. The antigenic determinant was designated virus membrane antigen (Vm). Further analyses revealed the presence of protein, lipid, and carbohydrate in a material having a molecular wt of about 6000 as determined by sodium dodecyl sulfate gel electrophoresis. Serological studies indicated that the outer membranes of AMV and other ChILSV are represented mainly by host cellular material.

- 5027 ANTIGENIC CHANGES IN TRANSFORMED FL HUMAN AMNION CELLS. (E.) Vladutiu, G. D. (Sch. Med., State U. New York, Buffalo) and N. R. Rose. *Clin Exp Immunol* 15(3):417-426, 1973.

During the normal cultivation of the long-established FL human amnion cell line the cells in one culture vessel became fibroblast-like, grew more rapidly than the original FL cells, and did not clump spontaneously as did the original cells. About 75% of the variant FL cells had nearly the diploid number of chromosomes (the remaining 25% had the normal heteroploid complement). However, the variant did not represent a reversion to the diploid ancestor in that it was lacking chromosomes, had several incomplete pairs, and did not have the time-limited growth capability characteristic of primary diploid amnion cells. The FL variant had two acid phosphatase isoenzymes and a rapidly migrating isoenzyme of glucuronidase which are not present in the normal cell, and a primate-specific cathodal esterase in the normal cell was absent in the variant. In addition, a glucose 6-phosphate dehydrogenase isoenzyme present in the normal FL was reduced in the variant, while an anodal isoenzymic form of esterase was increased in the variant. Further, both the enzymatic and antigenic determinants of the cathodal esterase isoenzyme were absent from the variant, although it was found to contain an anodal esterase isoenzyme which was antigenically unrelated to the cathodal esterase. The variant cells were human, possessed no mouse-specific antigens, were negative for mycoplasma infection, and showed no evidence of virus infection. It is concluded that the transformation of the FL amnion culture was spontaneous.

- 5028 AN IMMUNOFLUORESCENT STUDY OF TUMORS WITH SPECIFIC ANTISERA FOR SQUAMOUS EPITHELIAL INTERCELLULAR SUBSTANCE AND BASEMENT MEMBRANE. (E.) Pertschuk, L. P. (State U. New York, Downstate Med. Ctr., Brooklyn) and Y. Rosen. *Amer J Clin Pathol* 60(5):601-607, 1973.

Forty primary and six metastatic human neoplasms, principally of noncutaneous origin, were stained by

the immunofluorescent technique with antisera from cases of pemphigus specific for the intercellular substance of squamous epithelium (ICS). They were also stained with sera from patients with bullous pemphigoid which contained antibodies to squamous epithelial basement membrane. All 18 primary and metastatic squamous cell carcinomas showed positive ICS staining with the pemphigus sera. Of these tumors, 16 showed positive staining for basement membranes with the pemphigoid sera. In three tumors with epithelial "pearls", ICS and basement membrane substance were demonstrated within the "pearls". In general, there was poor correlation between histologic grading and degree of fluorescence in both primary and metastatic squamous cell carcinomas. Clear ICS staining was also seen in a pulmonary oat cell carcinoma, an undifferentiated jejunal carcinoma, a well differentiated colonic adenocarcinoma, and an alveolar cell carcinoma. No basement membrane substance was seen in any neoplasia other than those of unequivocal squamous cell origin.

- 5029 SPONTANEOUS MUTATION IN TISSUE CULTURE--CHEMICAL NATURE OF VARIANT IMMUNOGLOBULIN FROM MUTANT CLONES OF MOPC 21. (E.) Secher, D. S. (Med. Res. Council, Lab. Molecular Biol., Cambridge, England), R. G. H. Cotten and C. Milstein. *FEBS Lett* 37(2):311-316, 1973.

Clones from the mouse plasma cell tumor (MOPC 21) adapted to grow in tissue culture (P3K) were screened to reveal a variant clone (IF-2) which secreted a product of lower isoelectric point than the immunoglobulin (Ig) of the wild-type (P3K) line from which it was derived. The proteins of the IF-2 clone and a previously isolated variant clone (IF-1) secreting an Ig of altered isoelectric point contained light (L)-chains of normal size and heavy (H)-chains which differed from the P3K H-chain. The size difference between the IF-1 and wild-type H-chain was small. Although the IF-1 Ig contained sialic acid while the P3K Ig did not, this difference was not related to changes in the secretion process or other post-translational events. The mRNA's coding for the IF-1 and P3K H-chains were different, with the mutation involving an Ig structural gene. The IF-1 H-chain appeared to be a consequence of a deletion at the C-terminus, with valine as the new C-terminal residue. The size difference between the P3K and IF-2 H-chains was relatively great. The deletion in IF-2 appeared to be intramolecular, involving a region localized towards the N-terminal side of the hinge-region, which contains the inter-heavy disulfide bridges; this deletion could involve as many as 100 residues.

- 5030 CELLULAR REACTION IN TROPHOBLASTIC TUMOURS. (E.) Elston, C. W. (Kings Coll. Hosp. Med. Sch., London, England) and K. D. Bagshawe. (E.) *Br J Cancer* 28(3):245-256, 1973.

The presence of a mononuclear cell reaction to 41 gestational choriocarcinomas, 10 invasive moles and 13 malignant trophoblastic teratomas was investigated and the intensity of the reaction was graded. There

was a significantly better response to therapy and survival rate in those cases with a "severe" cellular reaction than in those with a "mild" reaction to gestational choriocarcinoma. The pathological and clinical features of invasive moles showed no relationship with the cellular reaction to the tumor. The cellular reaction to trophoblastic teratomas was generally poor although there was a marked cellular reaction to the tumor of one patient who experienced a sustained remission. The relationship of cellular reaction and response to treatment with other histological and clinical features was examined. With the exception of a positive correlation between the degree of vascular invasion and response to treatment, none was found. It is suggested that an infiltrate of mononuclear cells in gestational choriocarcinoma is probably a response to the presence of tumor antigens. The infiltrate favorably affects the response to chemotherapeutic agents, suggesting that it contributes to tumor cell death and it may be interpreted as an immunological response directed at tumor rejection.

- 5031 RELATION BETWEEN MIGRATION OF CELLS FROM PERITONEAL EXUDATE *IN VITRO* AND THE PRESENCE OF T LYMPHOCYTES. (Slov.) Mudzihradsky, J. (Inst. Exp. Oncol., Bratislava, Czechoslovakia) and F. Kalafut. *Biologia (Bratislava)* 28(3):161-164, 1973.
- 5032 IMMUNOGLOBULIN-POSITIVE REED-STERBERG CELLS IN HODGKIN'S DISEASE. (E.) Leech, J. (Sch. Med., Vanderbilt U., Nashville, Tenn.). *Lancet* (7823):265-266, 1973.
- 5033 A COLLABORATIVE STUDY OF A TEST FOR CARCINO-EMBRYONIC ANTIGEN (CEA) IN THE SERA OF PATIENTS WITH CARCINOMA OF THE COLON AND RECTUM. (E.) Natl. Cancer Inst. Canada Toronto, Ontario and American Cancer Soc. *Can Med Assoc J* 107(1):25-33, 1972.
- 5034 SNAIL AGGLUTININS IN TUMOR IMMUNOLOGY. (Ger.) Krüger, W. (Manfred von Ardenne Res. Inst., Dresden, Germany) and S. Schnitzler. *Dtsch Gesundheitsw* 27(28):1308-1311, 1972.
- 5035 HISTOCOMPATIBILITY ANTIGENS AND HEMATO-SARCOMAS. (Fr.) Moulinier, M. J. (Reg. Ctr. Blood Transfusion, Bordeaux, France), G. Hoerni-Simon, M. C. Merle and M. C. Mothey. *Bordeaux Med* 5(11):1285-1289, 1972.
- 5036 MULTIPLE MYELOMAS. II. BIOLOGY. (Fr.) Durand, M. M. (Bergonie Fdn., Bordeaux, France), E. Legrand and G. Hoerni-Simon. *Bordeaux Med* 5(11):1255-1261, 1972.
- 5037 EFFECT OF THE GRAFT VERSUS HOST REACTION ON GROWTH OF A SYNGENEIC SARCOMA IN MICE. (E.) Scherbakov, Va. Ya. (Smolensk Med. Inst., USSR Acad. Med. Sci., Moscow) and V. F. Semenov. *Bull Exp Biol Med* 75(2):164-167, 1973.
- 5038 IMMUNOMORPHOLOGICAL STUDY OF LOCALIZATION OF AZOCARCINOGEN-BINDING PROTEIN IN RAT LIVER SECTIONS. (E.) Bannikov, G. A. (Inst. Exp. Clin. Oncology, USSR Acad. Sci., Moscow) and T. A. Chipysheva. *Bull Exp Biol Med* 75(2):161-163, 1973.
- 5039 *IN VIVO* PROTECTION AGAINST MURINE PLASMA TUMOR GROWTH BY *IN VITRO* ACTIVATED SYNGENEIC LYMPHOCYTES. (E.) Röllinghoff, M. (Walter & Eliza Inst. Med. Res., Victoria, Australia) and H. Wagner. *J Natl Cancer Inst* 51(4):1317-1318, 1973.
- 5040 BLASTOGENIC TRANSFORMATION OF LYMPHOCYTES FOLLOWING PHYTOHAEMAGGLUTININ TREATMENT *IN VITRO* IN MALIGNANT LYMPHOMAS. II. RETICULOSARCOMA. (CORRELATION WITH THE IMMUNITY REACTION OF THE CELLULAR TYPE AND THE CLINICAL STATUS). (E.) Libansky, J. (Inst. Haematology, Blood Transfusion, Prague, Czechoslovakia), M. Sedlackova and I. Pinosova. *Neoplasma* 20(1):61-67, 1973.
- 5041 BLASTOGENIC TRANSFORMATION OF LYMPHOCYTES FOLLOWING PHYTOHAEMAGGLUTININ TREATMENT *IN VITRO* IN MALIGNANT LYMPHOMAS. III. CHRONIC LYMPHATIC LEUKAEMIA. (CORRELATION WITH IMMUNITY RESPONSE OF THE CELLULAR TYPE AND WITH THE CLINICAL STATUS). (E.) Libansky, J. (Inst. Hematology, Blood Transfusion, Prague, Czechoslovakia), M. Lukasova and I. Pinosova. *Neoplasma* 20(1):69-77, 1973.
- 5042 MULTIPLE DERMATOFIBROMAS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS ON IMMUNOSUPPRESSIVE THERAPY. (E.) Newman, D. M. (Toronto Gen. Hosp., Ontario, Canada) and J. B. Walter. *N Engl J Med* 289(16):842-843, 1973.
- 5043 ROSETTE-FORMING LYMPHOCYTES IN HODGKIN'S DISEASE. (E.) Cohnen, G. (U. Med. Ctr., Essen, Germany), W. Augener, G. Brittinger and S. D. Douglas. *N Engl J Med* 289(16):863, 1973.
- 5044 INFLUENCE OF VINBLASTINE ON ANTITUMOUR IMMUNITY IN MICE. (E.) Klener, P. (Charles U. Hosp., Prague, Czechoslovakia), J. Bubenik and J. Jandajsek. *Neoplasma* 20(3):239-242, 1973.
- 5045 EFFECT OF MYCOBACTERIUM BOVIS BCG-PRAHA ON THE GROWTH OF TRANSPLANTED DAEL'S SARCOMA IN GUINEA-PIGS. (E.) Schwartz, E. (Inst. Tuberculosis, Respiratory Diseases, Podunajske Biskupice, Czechoslovakia). *Neoplasma* 20(4):375-386, 1973.

- 5046 SUPPRESSION OF MURINE TUMOR GROWTH BY IMMUNE REACTION TO THE BACILLUS CALMETTE-GUERIN STRAIN OF *MYCOBACTERIUM BOVIS*. (E.) Bartlett, G. L. (Natl. Cancer Inst., Bethesda, Md.), B. Zbar and H. J. Rapp. *J Natl Cancer Inst* 48(1):245-258, 1972.
- 5047 THE POLYPEPTIDE STRUCTURE OF IMMUNOGLOBULIN: EXPERIMENTAL STUDIES ON THE RE-COMBINATION OF RADIOLABELLED MYELOMA POLYPEPTIDE CHAINS. (E.) Wetter, O. (Hlth. High Sch., Essen, Germany). *Z Klin Chem Klin Biochem* 11(9):388-392, 1973.
- 5048 SOME IMMUNOLOGICAL PROPERTIES OF LYMPHOID CELLS FROM PATIENTS WITH ACUTE LYMPHATIC LEUKAEMIA (ALL). (E.) Melief, C. J. M. (Netherlands Red Cross Blood Transfusion Service, Amsterdam), M. Schweitzer, W. P. Zeylemaker, E. H. Verhagen and V. P. Eijssvoogel. *Clin Exp Immunol* 15(1):131-143, 1973.
- 5049 CELLULAR IMMUNE RESPONSE AGAINST TUMOR CELLS. I. *IN VITRO* IMMUNIZATION OF ALLOGENEIC AND SYNGENEIC MOUSE SPLEEN CELL SUSPENSIONS AGAINST DBA MASTOCYTOMA CELLS. (E.) Lundak, R. L. (Sch. Med., U. California, Irvine) and D. J. Raidt. *Cell Immunol* 9(1):60-66, 1973.
- 5050 SOME APPROACHES TO MAPPING THE REDISTRIBUTION OF PARENT-CELL H-2 ANTIGENS ON THE MEMBRANE OF CELL HYBRIDS. (E.) Lengerova, A. (Inst. Experimental Biol., Genetics, Czechoslovak Acad. Sci., Prague) and J. Peknicova. *Eur J Cancer* 9(7):471-475, 1973.
- 5051 INHIBITION OF GRANULOPOIESIS BY ENDOGENOUS GRANULOCYTE CHALONE STUDIED WITH THE DIFFUSION CHAMBER TECHNIQUE. (E.) Vilpo, J. A. (2nd Dept. Path., U. Helsinki, Finland), K. Kiviniemi and T. Rytömaa. *Eur J Cancer* 9(7):515-524, 1973.
- 5052 IMMUNOSPECIFIC REGRESSION OF VARIOUS SYNGENEIC MOUSE TUMORS IN RESPONSE TO NEURAMINIDASE-TREATED TUMOR CELLS. (E.) Rios, A. (Dept.) Surg., U. Minnesota, Minneapolis) and R. L. Simmons. *J Natl Cancer Inst* 51(2):637-644, 1973.
- 5053 STUDIES ON THE COLUMN FRACTIONATION OF IMMUNE CELLS. III. TUMOR NEUTRALIZATION TEST. (Ger.) Eckert, R. (Central Inst. Cancer Res., Acad. Sci. DDR, Berlin-Buch, Germany) and G. Pasternak. *Acta Biol Med Ger* 31(2):305-310, 1973.
- 5054 GAMMOPATHY IN HAMSTERS WITH XENOGENEIC TUMOURS. (E.) Richman, A. V. (Wistar Inst. Anatomy, Biol., U. Pennsylvania, Philadelphia) and V. Defendi. *Nature [New Biol]* 245(144):158-160, 1973.
- 5055 SPECTROPHOTOMETRIC CHARACTERIZATION OF TISSUE EXTRACTS AND COLUMN ELUATES IN THE ISOLATION OF CARCINOEMBRYONIC ANTIGEN. (Ger.) Grossman, H. (Carl Gustav Carus Med. Acad., Dresden, Germany), G. Heidl and M. Müller. *Acta Biol Med Ger* 31(2):321-325, 1973.
- 5056 IMMUNOGLOBULIN RECEPTORS ON HUMAN LEUKOCYTES. IV. DIFFERENCES BETWEEN BONE MARROW AND BLOOD CELLS IN MULTIPLE MYELOMA AND CHRONIC LYMPHOCYTIC LEUKEMIA: EFFECTS OF THERAPY. (E.) Abdou, N. L. (VA Hosp., Philadelphia, Pa.) and N. I. Abdou. *J Lab Clin Med* 82(4):611-618, 1973.
- 5057 IMMUNOSUPPRESSION VERSUS IMMUNITY IN SYNGENEIC AND ALLOGENEIC TUMOURS. (E.) Vlachov, K. (Inst. Experimental Biol., Genetics, Czechoslovak Acad. Sci., Prague) and K. Nouza. *Neoplasma* 20(1):3-12, 1973.
- 5058 BLASTOGENIC TRANSFORMATION OF LYMPHOCYTES FOLLOWING PHYTOHAEMAGGLUTININ TREATMENT *IN VITRO* IN MALIGNANT LYMPHOMAS. I. HODGKIN'S DISEASE. (CORRELATION WITH IMMUNITY RESPONSE OF THE CELLULAR TYPE AND WITH THE CLINICAL STATUS). (E.) Libansky, J. (Inst. Haematology, Blood Transfusion, Prague, Czechoslovakia), M. Lukasova and I. Pinosova. *Neoplasma* 20(1):51-60, 1973.
- 5059 LYMPHOMA-ASSOCIATED AND HL-A ANTIGENS IN THE MIXED LEUKOCYTE REACTIVITY BETWEEN IDENTICAL SIBLINGS. (E.) Mavligit, G. M. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston), J. U. Gutterman, E. M. Herish, R. D. Rossen, W. T. Butler, K. B. McCredie and E. J. Freireich. *Transplantation* 16(3):217-220, 1973.
- 5060 IMMUNOABSORBENT PURIFICATION OF ANTISERA TO IMPURE ABNORMAL SERUM ANTIGENS. APPLICATION TO MURINE LEUKEMIA PLASMA VIRUS. (E.) Weliky, N. (Systems Group of TRC Inc., Redondo Beach, Calif.), B. J. Kallman and D. H. Leaman. *Immunol Commun* 2(3):297-302, 1973.
- 5061 HIGH FREQUENCY OF SPONTANEOUS LYMPHOBLASTOID TRANSFORMATION *IN VITRO* CULTURE OF LEUKOCYTES FROM HUMAN LYMPH NODES AND PERIPHERAL BLOOD. (E.) Takada, M. (Dept. Virology, Kitasato Inst., Tokyo, Japan). *Jcp J Exp Med* 43(4):341-348, 1973.
- 5062 FLUCTUATIONS IN THE TITRE OF ANTIBODY TO A SOLUBLE ANTIGEN OF MYXOMA VIRUS IN FIELD POPULATIONS OF RABBITS, *ORYCTOLAGUS CUNICULUS* (L.), IN AUSTRALIA. (E.) Williams, R. T. (Wildlife Res., Canberra, Australia), J. D. Dunsmore and W. R. Sobey. *J Hyg (Camb)* 71(3):487-500, 1973.

- 5063 DEGRADATION OF IMMUNOGLOBULINS BY LYSOSOMAL ENZYMES OF TUMORS - I. DEMONSTRATION OF THE PHENOMENON USING MOUSE TUMORS. (E.) Keisari, Y. (Dept. Microbiol., Tel Aviv U., Israel) and I. P. Witz. *Immunochemistry* 10(9):565-570, 1973.
- 5064 TUMOR TRANSPLANTATION, CELL NUMBER, AND MITOTIC RHYTHMS IN LOW DOSE CHALLENGES BY EHRlich ASCITES TUMOR. (E.) Brown, H. R. (U. Arkansas Med. Ctr., Little Rock) and E. R. Burns. *Transplantation* 16(3):199-204, 1973.
- 5065 HETEROTRANSPLANTATION OF HUMAN MALIGNANT TUMORS IN "NUDE" THYMUSLESS MICE. I. BREEDING AND MAINTENANCE OF "NUDE" MICE. (E.) Giovannella, B. C. (St. Joseph Hosp., Houston, Tex.) and J. S. Stehlin. *J Natl Cancer Inst* 51(2):615-619, 1973.
- 5066 IMMUNOLOGICAL REACTION IN KERATOACANTHOMA, A SPONTANEOUSLY RESOLVING SKIN TUMOR. (E.) Brown, F. C. (U.S. Navy Hosp., San Diego, Calif.) and E. M. Tan. *Cancer Res* 33(9):2030-2033, 1973.
- 5067 EVALUATION OF THE USEFULNESS OF PLASMA LEVELS OF CARCINOEMBRYONIC ANTIGEN IN THE DETECTION OF CANCER AND THE MONITORING OF CANCER THERAPY. (E.) Langan, J. (Temple U. Med. Sch., Philadelphia, Pa.), F. Demyan and C. Sims. *Lab Med* 4(9):27-30, 1973.
- 5068 CUTANEOUS DELAYED HYPERSENSITIVITY RESPONSES TO TUMOR-ASSOCIATED AND OTHER ANTIGENS IN ACUTE LEUKEMIA. (E.) Char, D. H. (Natl. Cancer Inst., Bethesda, Md.), A. Lepourhiet, B. G. Leventhal and R. B. Herberman. *Int J Cancer* 12(2):409-419, 1973.
- 5069 IMMUNE DEFICIENCIES AND KAPOSI'S SARCOMA. (E.) Dobozy, A. (U. Med. Sch., Szeged, Hungary), S. Husz, J. Hunyadi, G. Berko and N. Simon. *Lancet* (7829):625, 1973.
- 5070 GRAFT VERSUS LEUKEMIA. III. APPARENT INDEPENDENT ANTIHOST AND ANTILEUKEMIC ACTIVITY OF TRANSPLANTED IMMUNOCOMPETENT CELLS. (E.) Bortin, M. M. (Mount Sinai Med. Ctr., Milwaukee, Wisconsin), A. A. Rimm, E. C. Saltzstein and G. E. Rodey. *Transplantation* 16(3):182-188, 1973.
- 5071 T CELL SURFACE MARKERS ON LYMPHOBLASTS FROM ACUTE LYMPHOCYTIC LEUKEMIA. (E.) Borella, L. (St. Jude Children's Res. Hosp., Memphis, Tenn.) and L. Sen. *J Immunol* 111(1):1257-1260, 1973.
- 5072 MACROPHAGES AND THE TUMOUR BEARING HOST. (E.) Evans, R. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England). *Br J Cancer* 28(Suppl.1):19-25, 1973.
- 5073 EXPRESSION OF TUMOR-SPECIFIC ANTIGENS IN MOUSE SOMATIC CELL HYBRIDS. (E.) Jami, J. (Inst. Sci. Cancer Res., Natl. Sci. Res. Ctr., Villejuif, France) and E. Ritz. *Cancer Res* 33(10):2524-2528, 1973.
- 5074 THYMUS FUNCTION IN SPONTANEOUS LYMPHOID LEUKEMIA. II. *IN VITRO* RESPONSE OF "PRE-LEUKEMIC" AND LEUKEMIC THYMUS CELLS TO MITOGENS. (E.) Nagaya, H. (Duke U. Med. Ctr., Durham, N.C.). *J Immunol* 111(4):1052-1060, 1973.
- 5075 EFFECT OF LOCALLY APPLIED CORTISOL ACETATE ON DELAYED HYPERSENSITIVITY REACTIONS IN CANCER PATIENTS. (E.) Al-Sarraf, M. (Division Oncology, Wayne St. U., Detroit, Mich.), S. Sardesai and V. K. Vaitkevicius. *Oncology* 28(2):97-103, 1973.
- 5076 IMMUNE RESPONSE TO A SYNGENEIC RAT TUMOUR: DEVELOPMENT OF REGIONAL NODE LYMPHOCYTE ANERGY. (E.) Flannery, G. R. (Monash U. Med. Sch., Melbourne, Victoria, Australia), P. J. Chalmers, J. M. Rolland and R. C. Nairn. *Br J Cancer* 28(2):118-122, 1973.
- 5077 THE EFFECT OF LETHALLY IRRADIATED CELLS ON THE TRANSPLANTABILITY OF MURINE TUMOURS. (E.) Hewitt, H. B. (Mount Vernon Hosp., Northwood, Middlesex, England), E. Blake and E. H. Porter. *Br J Cancer* 28(2):123-135, 1973.
- 5078 STRUCTURE AT 4.5 Å RESOLUTION OF A PHOSPHORYLCHOLINE-BINDING FAB. (E.) Padlan, E. A. (Natl. Inst. Hlth., Bethesda, Md.), D. M. Segal, T. F. Spande, D. R. Davies, S. Rudikoff and M. Potter. *Nature [New Biol]* 245(145):165-167, 1973.
- 5079 DIFFERENTIATION OF T CELLS INDUCED BY PREPARATIONS FROM THYMUS AND BY NONTHYMIC AGENTS. THE DETERMINED STATE OF THE PRECURSOR CELL. (E.) Scheid, M. P. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), M. K. Hoffman, K. Komuro, U. Hämmerling, J. Abbott, E. A. Boyse, G. H. Cohen, J. A. Hooper, R. S. Schulof and A. L. Goldstein. *J Exp Med* 138(4):1027-1032, 1973.
- 5080 SOME CHARACTERISTICS OF IMMUNOGLOBULIN INVOLVED IN ANTIBODY DEPENDENT LYMPHOCYTE CYTOTOXICITY. (E.) MacLennan, I. C. M. (Radcliffe Infirmary, Oxford, England) and B. Harding. *Br J Cancer* 28(Suppl.1):7-10, 1973.
- 5081 INCIDENCE OF AUTOANTIBODIES IN CANCER PATIENTS. (E.) Tannenberg, A. E. G. (Monash U. Med. Sch., Melbourne, Australia), H. K. Muller, M. N. Cauchi and R. C. Nairn. *Clin Exp Immunol* 15(6):153-156, 1973.

- 5082 ENHANCEMENT OF THE IMMUNITY AGAINST THE TRANSPLANTED HEPATOMA 22A CELL CULTURE IN MICE. (Rus.) Korosteleva, T. A. (N.N. Petrov Res. Inst. Oncology, USSR Ministry Publ. Hlth., Leningrad), Ju. T. Alexanyan and K. S. Movsesyan. *Vopr Onkol* 19(8):72-76, 1973.
- 5083 INCREASED CYTOLYTIC EFFECT OF IMMUNE LYMPHOCYTES IN A SYNGENEIC TUMOUR SYSTEM FOLLOWING SIMPLE PURIFICATION PROCEDURES. (E.) Vasudevan, D. M. (Swiss Inst. Exp. Cancer Res., Lausanne), K. T. Brunner and J.-C. Cerottini. *Br J Cancer* 28(Suppl.1):35-36, 1973.
- 5084 ANTIGENIC SURFACE DETERMINANTS OF CHICKEN LYMPHOID CELLS. I. SEROLOGIC PROPERTIES OF ANTI-BURSA AND ANTI-THYMUS SERA. (E.) Wick, G. (Inst. Gen. Exp. Pathol., U. Vienna, Austria), B. Albin and F. Milgrom. *Clin Exp Immunol* 15(2):237-249, 1973.
- 5085 *IN VITRO* CYTOTOXICITY OF RABBIT AND GUINEA PIG ANTISERA AGAINST THE ZAJDELA ASCITES HEPATOMA OF RAT. (Ger.) Krüger, W. ("Manfred von Ardenne" Res. Inst., Dresden, German Democ. Repub.). *Acta Biol Med Ger* 30(6):863-874, 1973.
- 5086 THORACIC-DUCT AND BLOOD LYMPHOCYTES IN CANCER. (E.) Benninghoff, D. L. (Downstate Med. Ctr., Brooklyn, N.Y.), R. E. Girardet and D. D. Porteous. *Lancet* (7823):264-265, 1973.
- 5087 HUMORAL AUTOIMMUNE RESPONSE AGAINST RABBIT MALE ACCESSORY GLANDS ELICITED BY UNTREATED AND CHEMICALLY MODIFIED RABBIT SEMINAL PLASMA. (E.) Vottero-Cima, E. (Natl. U. Cordoba, Argentina), M. A. Vides and C. Yantorno. *Ann Immunol (Inst. Pasteur)* 124C(2):273-284, 1973.
- 5088 ANTIGEN-BINDING CELLS FOLLOWING CONTACT-SENSITIZATION DETECTED BY ROSETTE-FORMATION. CHARACTERIZATION OF THE RECEPTORS BY INHIBITION OF ROSETTE-FORMING CELLS WITH ANTI-RABBIT ALLOTYPE AND ANTI-CLASS SPECIFIC ANTISERA. (E.) Ferrarini, M. (Dept. Path., U. Cambridge, England), A. Munro, S. P. Kent, B. Gurner and R. R. A. Coombs. *Int Arch Allergy Appl Immunol* 44(3):321-337, 1973.
- 5089 IMMUNOLOGICAL STIMULATION WITH MODIFIED CANCER CELLS. (E.) Prager, M. D. (U. Texas, Southwestern Med. Sch., Dallas). *Biomedicine* 18(4):261-263, 1973.
- 5090 ISOLATION OF AN "ANTIGEN" FROM MALIGNANT TUMOURS. (E.) Carnegie, P. R. (Newcastle Gen. Hosp., Newcastle upon Tyne, England), E. A. Caspary and E. J. Field. *Br J Cancer* 28(Suppl.1):219-223, 1973.
- 5091 INHIBITION OF LEUCOCYTE MIGRATION BY TUMOUR-ASSOCIATED ANTIGENS OF THE COLON AND RECTUM. (E.) Guillou, P. J. (Leeds St. James's U. Hosp., England) and G. R. Giles. *Gut* 14(9):733-738, 1973.
- 5092 TRANSFER OF ANTIGEN FROM MACROPHAGES TO LYMPHOCYTES. II. IMMUNOLOGICAL SIGNIFICANCE OF THE TRANSFER OF LIPOPOLYSACCHARIDE. (E.) Bona, C. (Claude Bernard Hosp., Paris, France), R. Robineaux, A. Anteunis, C. Heuclin and A. Astesano. *Immunology* 24(5):831-840, 1973.
- 5093 TECHNICAL ASPECTS OF THE MACROPHAGE ELECTROPHORETIC MOBILITY (MEM) TEST FOR MALIGNANT DISEASE. (E.) Pritchard, J. A. V. (Velindre Hosp., Whitchurch, Cardiff, Wales), J. L. Moore, W. H. Sutherland and C. A. F. Joslin. *Br J Cancer* 28(Suppl.1):229-236, 1973.
- 5094 ASPECTS OF THE STRUCTURE AND CLINICAL ROLE OF THE CARCINOEMBRYONIC ANTIGEN (CEA) AND RELATED MACROMOLECULES WITH PARTICULAR REFERENCE TO UROTHELIAL CARCINOMA. (E.) Neville, A. M. (Roy. Cancer Hosp., London, England), R. Nery, R. R. Hall, C. Turberville and D. J. R. Laurence. *Br J Cancer* 28(Suppl.1):198-207, 1973.
- 5095 MACROPHAGE ELECTROPHORETIC MOBILITY (MEM) TEST IN CANCER: A CRITICAL EVALUATION. (E.) Field, E. J. (Newcastle Gen. Hosp., Newcastle upon Tyne, England), E. A. Caspary and K. S. Smith. *Br J Cancer* 28(Suppl.1):208-214, 1973.
- 5096 MACROPHAGE ELECTROPHORETIC MIGRATION (MEM) TEST FOR LYMPHOCYTE SENSITIZATION: SOME PRACTICAL EXPERIENCES IN MACROPHAGE SELECTION. (E.) Shenton, B. K. (Newcastle Gen. Hosp., Newcastle upon Tyne, England), D. Hughes and E. J. Field. *Br J Cancer* 28(Suppl.1):215-218, 1973.
- 5097 CLINICAL EVALUATION OF THE MAKARI TUMOUR SKIN TEST. (E.) Tee, D. E. H. (King's Coll. Hosp. Med. Sch., London, England). *Br J Cancer* 28(Suppl.1):187-197, 1973.
- 5098 INFLUENCE OF GENOTYPE OF HOST ON REGRESSION OF SOLID AND ASCITIC FORMS OF SARCOMA 180 AND EFFECT OF CHEMOTHERAPY ON THE SOLID FORM. (E.) Tarnowski, G. S. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), I. M. Mountain and C. C. Stock. *Cancer Res* 33(8):1885-1888, 1973.
- 5099 ALPHA-CHAIN DISEASE IN SUBSAHARAN AFRICA. (E.) Novis, B. H. (U. Cape Town Med. Sch., S. Africa), L. B. Kahn and S. Bank. *Am J Dig Dis* 18(8):679-688, 1973.

- 5100 THE SIGNIFICANCE OF HUMORAL ANTIBODIES IN THE LOCALIZATION OF HUMAN MALIGNANT MELANOMA. (E.) Lewis, M. G. (Mem. U. Newfoundland, St. John's, Canada), E. McCloy and J. Blake. *Br J Surg* 60(6):443-446, 1973.
- 5101 IMMUNE RESPONSE TO SHEEP RED CELLS IN AKR MOUSE LEUKEMIA. (E.) Ram, M. D. (Case Western Reserve U., Cleveland, Ohio), Kohn, R. R. and D. Novak. *Am J Pathol* 72(1):39-52, 1973.
- 5102 IMPAIRED LYMPHOCYTE TRANSFORMATION IN HODGKIN'S DISEASE. EVIDENCE FOR DEPLETION OF CIRCULATING T-LYMPHOCYTES. (E.) Matchett, K. M. (Duke U. Med. Ctr., Durham, N.C.), A. T. Huang and W. B. Kremer. *J Clin Invest* 52(8):1908-1917, 1973.
- 5103 THE PURIFICATION OF MEMBRANE-ASSOCIATED TUMOUR ANTIGENS BY PREPARATIVE POLYACRYLAMIDE GEL ELECTROPHORESIS. (E.) Harris, J. R. (U. Nottingham, England), M. R. Price and R. W. Baldwin. *Biochem Biophys Acta* 311(4):600-614, 1973.
- 5104 ANTIIMMUNOGENIC EFFECT OF SPECIFIC ANTIBODY ON THE MIXED LYMPHOCYTE REACTION. (E.) Milton, J. D. (St. Mary's Hosp. Med. Sch., London, England), J. F. Mowbray, M. Ruzsiewicz and C. B. Carpenter. *Transplantation* 15(6):579-585, 1973.
- 5105 INFLUENCE OF TUMOUR GROWTH ON THE EVOLUTION OF CYTOTOXIC LYMPHOID CELLS IN RATS BEARING A SPONTANEOUSLY METASTASIZING SYNGENEIC FIBROSARCOMA. (E.) Currie, G. A. (Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England) and J. O. Gage. *Br J Cancer* 28(2):136-146, 1973.
- 5106 CARCINOEMBRYONIC ANTIGEN IN PATIENTS WITH CARCINOMA OF THE LUNG. (E.) Vincent, R. G. (Roswell Park Mem. Inst., Buffalo, N.Y.) and T. M. Chu. *J Thorac Cardiovasc Surg* 66(2):320-328, 1973.
- 5107 HEPATOMA WITH SERUM ALPHA-1-FETOPROTEIN WHICH SIMULATED SECONDARY HEPATIC CARCINOSIS. (Fr.) Chaput, J. C. (Antoine Beclere Hosp., Clamart, France), B. Rain, P. H. Cassan and E. Martin. *Nouv Presse Med* 2(31):2057, 1973.
- 5108 DETECTION OF ANTIGENS IN VIRAL-INDUCED MAMMARY TUMORS IN MOUSE LINE CBA/B₁. (Rus.) Zotter, S. (Carl Gustav Carus Med. Acad., Dresden, East Germany), M. Müller, K. Munschner and S. Liebscher. *Vopr Onkol* 19(9):60-67, 1973.
- 5109 EARLY DETECTION OF CANCER. (Fr.) Burtin, P. (No affiliation). *Recherche* (37):796-797, 1973.
- 5110 ERYTHROCYTE MEMBRANE DEFECTS ASSOCIATED WITH A TRANSPLANTABLE LYMPHOID TUMOR. (E.) Chandler, F. W., Jr. (Coll. Vet. Med., U. Georgia, Athens) and O. J. Fletcher, Jr. *J Natl Cancer Inst* 51(4):1351-1353, 1973.
- 5111 YOLK SAC TUMOR (ENDODERMAL SINUS TUMOR) AND ALPHA-FETOPROTEIN. A REPORT OF THREE CASES. (E.) Tsuchida, Y. (Depts. Pediatric Surg., Pediatrics, Path., Internal Med., U. Tokyo, Japan), S. Saito, M. Ishida, K. Ohmi, Y. Urano, Y. Endo and T. Oda. *Cancer* 32(4):917-921, 1973.
- 5112 IMMUNOLOGIC PARAMETERS IN HISTIOCYTOSIS-X. (E.) Leikin, S. (Children's Hosp., Washington, D.C.), G. Puruganan, A. Frankel, R. Steerman and R. Chandra. *Cancer* 32(4):796-802, 1973.
- 5113 CELL SURFACE ALTERATIONS IN TRANSFORMED AND MITOTIC NORMAL CELLS MONITORED WITH PLANT AGGLUTININS. (E.) Burger, M. M. (Bioctr. U. Basel, Switzerland). *Neoplasma* 20(5):579-581, 1973.
- 5114 AUTOIMMUNE HEMOLYTIC ANEMIA TERMINATING SEVEN YEARS LATER IN HODGKIN'S DISEASE. (E.) Cazenave, J.-P. (Dept. Path., McMaster U., Hamilton, Ontario, Canada), J. A. E. Gagnon, E. Girouard and A. Bastarache. *Can Med Assoc J* 109(8):748-752, 1973.
- 5115 LYMPHOCYTE TRANSFORMATION IN MALIGNANT LYMPHOMAS. (E.) Greally, J. (U. Minnesota Hosp.), I. E. Fortuny and E. J. Yunis. *Ir J Med Sci* 142(5):255-262, 1973.
- 5116 STIMULATION OF DNA SYNTHESIS IN MOUSE LYMPHOID CELLS BY POLYANIONS *IN VITRO*. I. TARGET CELLS AND POSSIBLE MODE OF ACTION. (E.) Diamantstein, T. (Steglitz Clin., Free U. Berlin, Germany), W. Vogt, H. Rühl and G. Bochart. *Eur J Immunol* 3(8):488-493, 1973.
- 5117 IMMUNOCHEMICAL STUDY OF TISSUE α_2 -GLOBULIN IN TUMOR AND NORMAL TISSUE OF HUMAN KIDNEY. (Rus.) Prokopenko, P. G. (2nd Moscow N.I. Pirogov Med. Inst., U.S.S.R.) and Yu. S. Tatarinov. *Biull Eksp Biol Med* 76(8):102-105, 1973.
- 5118 TUMOR IMMUNITY INDUCED BY BCG-TUMOR CELL MIXTURES IN SYNGENEIC MICE. (E.) Tokunaga, T. (Natl. Inst. Hlth., Tokyo, Japan), T. Kataoka, R. M. Nakamura, S. Yamamoto and T. Tanaka. *Jap Med Sci Biol* 26(2):71-85, 1973.
- 5119 ⁵¹Cr RELEASE MICROASSAYS FOR HISTOCOMPATIBILITY ANTIGENS ON NORMAL AND MALIGNANT CELLS. (E.) Anonymous. *Transplantation* 16(3):246-250, 1973.

5120 STIMULATION OF DNA SYNTHESIS IN MOUSE
LYMPHOID CELLS BY POLYANIONS *IN VITRO*.
II. RELATIONSHIP BETWEEN ADJUVANT ACTIVITY AND
STIMULATION OF DNA SYNTHESIS BY POLYANIONS. (E.)
Vogt, W. (Steglitz Clin., Free U. Berlin, Germany),
H. Rühl, B. Wagner and T. Diamantstein. *Eur J Immunol*
3(8):493-496, 1973.

5121 QUANTITATIVE STUDIES OF MACROPHAGES IN
BLOOD CULTURES IN CHRONIC LYMPHOCYTIC
LEUKAEMIA. (E.) Navone, R. (Inst. Anatomy, Path.
Histology, U. Torino, Turin, Italy), G. Mazzucco
and A. Stramignoni. *Acta Haematol* 49(6):335-339,
1973.

See also:

- * (Rev): 4809
- * (Chem): 4826, 4833, 4848, 4851, 4861
- * (Viral): 4935, 4938, 4976, 4980, 4981

- 5122 ACHLORHYDRIA, CHRONIC ATROPHIC GASTRITIS AND STOMACH CANCER. (Sp.) Freytes, M. A. (San Roque Hosp., Cordoba, Argentina) and J. H. Carri. *Acta Gastroenterol Latinoam* 5(2):61-64, 1973.

Hypochlorhydria (maximal acid secretion of less than 5 mEq/hr) or achlorhydria was diagnosed in 329 of 3158 patients by the augmented histamine test. These patients consisted of 198 women and 131 men, aged 15-81 yr (mean age 54 yr). Gastric biopsies, performed on 387 patients, revealed that 117 (74 women and 43 men, aged 14-80 yr; mean age 47 yr) had chronic atrophic gastritis. Of these 117 patients, 48 (41%) had achlorhydria, 60 (51%) had hypochlorhydria, and 9 (8%) had normochlorhydria. Gastric adenocarcinomas were diagnosed in 13 (11%) of the patients with chronic atrophic gastritis. Gastric biopsies showed that adenocarcinomas were present in 33 of the 387 biopsied patients. Of these 33 patients (22 men and 11 women, aged 32-78 yr; mean age 58 yr), 23 (70%) had achlorhydria and 10 (30%) had hypochlorhydria. The high incidence of gastric cancer in patients with achlorhydria and/or chronic atrophic gastritis suggests that these patients should be followed for early detection of stomach cancer.

- 5123 THE PRECURSORS OF ENDOMETRIAL CANCER: A STUDY OF THEIR CELLULAR MANIFESTATIONS. (E.) Ng, A. B. P. (Inst. Path., Case Western Reserve U., Cleveland, Ohio), J. W. Reagan and R. L. Cechner. *Acta Cytol (Baltimore)* 17(5):439-448, 1973.

Cell samples prepared from endocervical aspirations obtained from 116 women with proven endometrial hyperplasia and adenocarcinoma *in situ* were evaluated. Of these 36 were classified as cystic hyperplasia, 41 as adenomatous hyperplasia, 28 as atypical hyperplasia, and 11 as adenocarcinoma *in situ* of the endometrium. The distribution of the abnormal cells, nuclear and cytoplasmic characteristics and the nature of the host response were evaluated. The observed cellular abnormalities include cellular and nuclear enlargement, nuclear hyperchromasia, alterations in nuclear chromatin and changes in the nucleolus. The degree of the abnormalities is related to the severity of the endometrial abnormality. The altered cells are often accompanied by erythrocytes and evidence of an estrogenic effect while the milieu associated with endometrial cancer is not observed in endometrial hyperplasia and is present in less than 20 percent of the cases with adenocarcinoma *in situ*. When the cellular characteristics of endometrial hyperplasia, adenocarcinoma *in situ*, and Grade I adenocarcinoma are compared, differences become apparent. In endocervical cell samples, cells derived from cystic and adenomatous hyperplasia of the endometrium more closely resemble those of normal endometrial cells. Conversely, to some degree the cellular abnormalities more nearly resemble those associated with Grade I endometrial adenocarcinoma. These observations are in keeping with the overall trends in endometrial carcinogenesis. Simple collection methods provide only spontaneously desquamated cells which are inconstant in their occurrence. Samples obtained from the uterine cavity

provide a more constant and a greater concentration of characteristic cells and may prove to be more suitable for the detection of endometrial hyperplasia.

- 5124 A STUDY ON MORPHOLOGICAL CHANGES IN THE TESTES OF OLD ALBINO RATS. (E.) Lützen, L. (Dept. Exp. Pathol., Dr. Karl Thomae, GmbH, Biberach/Riss, W. Germany) and H. Ueberberg. *Beitr Pathol* 149(4):377-385, 1973.

The testes of 90, 2 yr old male SPF-albino rats were examined. String-like hyperplasia of the Leydig cells were found in 27 of the animals. Nodular or adenomatous proliferation in one or both testes was found in 49. Some lesions were microscopic in size, others occupied the entire testis. These were named adenomas of the Leydig cells. No malignant lesions were found. These changes have not been found in animals up to 1 yr of age. It is suggested that these adenomata are initially due to a process started by the pituitary and that their growth became autonomous. Atrophic changes in the germinal epithelium were also found, but these do appear in younger animals in the same form.

- 5125 ATYPICAL NUCLEAR INVAGINATION IN LYMPHOBLASTIC LYMPHOSARCOMA. (Ger.) Kaiserling, E. (Pathol. Inst., U. Kiel, Germany) and H. Stein. *Virchows Arch [Zellpathol]* 13(3):215-225, 1973.

- 5126 INTRACRANIAL HYPERPLASIA OF SPENOETHMOID CELLS AS A SIGN OF A MENINGIOMA. (Ger.) Wiggli, U. (Cantonal Hosp., U. Lausanne, Switzerland) and R. Oberson. *Schweiz Med Wochenschr* 103(43):1492-1499, 1973.

- 5127 LOCALIZATION OF THE ONSET OF INVASIVE CANCER GROWTH IN THE CERVIX. (Ger.) Pickel, H. (Clin. Gynecol. Obstet., U. Graz, Austria) and E. Burghardt. *Arch Gynaekol* 215(2):189-198, 1973.

- 5128 IS THERE SURFACE GROWTH IN INTRAEPITHELIAL CARCINOMAS OF THE CERVIX? (Ger.) Burghardt, E. (Clin. Gynecol. Obstet., U. Graz, Austria). *Arch Gynaekol* 215(1):1-16, 1973.

- 5129 FORMATION OF TUMORS IN LEUKOSES. (Ger.) Löhr, J. (Pathol. Inst., Johannes Gutenberg U., Mainz, Germany) and K. Hill. *Blut* 27(2):81-91, 1973.

- 5130 MORPHOLOGICAL CHARACTERISTICS OF STOMACH CANCER IN FINDINGS FROM THE GLIWICE ONCOLOGICAL INSTITUTE. (Ger.) Podworski, H. (Oncol. Inst., Gliwice, Poland). *Arch Geschwulstforsch* 41(1):23-33, 1973.
- 5131 HISTOGENESIS OF SCIRRHUS CARCINOMA OF THE HUMAN BREAST. (E.) Evgen'eva, T. P. (A. N. Severtsov Inst. Evolutionary Morphology, Ecology Animals, USSR Acad. Sci., Moscow). *Bull Exp Biol Med* 74(12):69-71, 1972.
- 5132 SO-CALLED ARACHNOIDAL SARCOMA OF THE CEREBELLUM: ITS HISTOGENESIS AND CLINICAL COMPARISON WITH CLASSICAL MEDULLOBLASTOMA. (Jap.) Soejima, T. (Fac. Med., Kyushu U., Fukuoka, Japan), M. Fukui and K. Kitamura. *Brain Nerve (Tokyo)* 25(10):1275-1283, 1973.
- 5133 BONE JOINT CHANGES PRECEDING THE DEVELOPMENT OF ACUTE MYELOBLASTIC LEUKEMIA. (Pol.) Zimmermann-Gorska, I. (J. Strus Hosp., Poznan, Poland) and E. Celinska-Szpytko. *Rewmatologia* 10(2):165-169, 1972.
- 5134 MORPHOLOGY AND HISTOGENESIS OF RETICULOHISTIOCYTOMA. (Rus.) Apatenko, A. K. (Central Dept. Military Med., Min. Defense USSR) *Vopr Onkol* 19(8):61-68, 1973.
- 5135 STRAIN-LINKED PATTERNS IN THE EPIDERMIS DIRECTLY CORRELATABLE TO EVOLUTION OF PRIMARY MULTIPLE SKIN TUMOURS IN MICE. A THEORY OF SKIN-TUMOUR EVOLUTION. (E.) Setälä, K. (1st Dept. Path., U. Helsinki, Finland), L. Stjernvall, I. Schreck-Purola and T. Telaranta. *Vth Perugia Quadrennial Intl Conference on Cancer* 46, 1973.
- 5136 FOCAL VERSUS SYSTEMIC DEVELOPMENT OF LYMPHORETICULAR NEOPLASMS. (E.) Kreuger, G. R. F. (Path. Inst., U. Cologne, Germany). *Vth Perugia Quadrennial Intl Conference on Cancer* 108, 1973.
- 5137 HYPOTHALAMIC-HYPOPHYSEAL RELATIONSHIPS IN INDUCTION OF THYROTROPIC PITUITARY TUMORS: FURTHER STUDIES. (E.) Clifton, K. H. (U. Wisconsin Med. Sch., Madison) and M. B. Yatvin. *Vth Perugia Quadrennial Intl Conference on Cancer* 105, 1973.
- 5138 MULTIPLE ENDOCRINE ADENOMAS (PLURIGLANDULAR SYNDROME) IN EXPERIMENTAL ANIMALS AND IN MAN. A HISTOPATHOLOGIC CONSIDERATION AND PATHOGENESIS. (E.) Berdjis, C. C. (Armed Forces Inst. Path., Washington, D.C.). *Vth Perugia Quadrennial Intl Conference on Cancer* 101, 1973.

See also:

- * (Rev): 4801
- * (Chem): 4834
- * (Viral): 4936, 4944
- * (Immun): 5004, 5074

- 5139 MALIGNANT MORTALITY IN NORTH AND SOUTH DAKOTA: 1950-1967. (E.) Frigerio, N. A. (Nat'l. Coll., Lombard, Ill.) and J. R. Wutzke. *S Dakota J Med* 26(11):33-37, 1973.

Malignant rates in North and South Dakota were abstracted and analyzed for the 18 yr period, 1950-1967, in an effort to aid physicians in their attempts to diagnose the probable malignancies of their patients living in this area. Both states have rates lower than the U. S. as a whole in the buccal and pharynx category, in the digestive organ category (except the stomach), in respiratory sites, in breast cancer among females, in female organ sites (except the uterus and ovary and the significance here was not great), and in the skin and integument. Males in North Dakota have elevated rates for the small intestine and peritoneum and prostate carcinoma. In the urinary tract the pattern of malignant rates is mixed. The average change in malignant mortality rate over the 18 yr period showed that Dakota rates were either decreasing more rapidly than the U. S. rates as a whole, or, if increasing, were increasing more slowly. It is suggested that altitude, radiation background, or lack of urbanization may be important factors in producing the low level of malignant mortality.

- 5140 COLONIC AND STOMACH CANCERS: OPPOSING AETIOLOGIES? (E.) Howell, M. A. (Nat'l. Cancer Inst., Bethesda, Md.). *Lancet* (7841):1338, 1973.

The reportedly consistent negative relationship between colonic cancer and stomach cancer has been interpreted as indicating opposing etiologies. However, death rates by state within the United States and by prefecture within Japan yield significant positive correlations between colonic and stomach cancer, and data based on the death rates from these cancers in 41 countries indicate no significant correlation. A similar lack of correlation is found in the data reporting the international incidence of these cancers among males. Thus, since there is no clear evidence of a causal connection between colonic and stomach cancer, the theory of opposing etiologies cannot be substantiated.

- 5141 OVARIAN CANCERS IN THE YOUNG. EPIDEMIOLOGIC OBSERVATIONS. (E.) Li, F. P. (Child. Cancer Res. Fdn., Boston Mass.), J. F. Fraumeni, Jr. and N. Dalager. *Cancer* 32(4):969-972, 1973.

Study was made of U.S. mortality rates for ovarian cancers among 1,135 girls aged 0-19 yr, 1950-1968, and of 59 hospital records for children with these neoplasms. The death rates showed no significant changes over the 18-yr period. Ovarian cancer mortality was low in young children, and the increase during adolescence and early adulthood was about 30% higher among nonwhites. Of the 258 ovarian tumors typed on the death certificates of persons under 20 yr of age, 153 (59%) were of the

germ-cell type (teratocarcinoma, dysgerminoma, and embryonal carcinoma) in contrast to a predominance of cystadenocarcinomas which occurs in older persons. Gonadal dysgenesis was the only congenital defect associated with ovarian neoplasia. There was no evidence of unusual exposure to estrogen hormones or anticonvulsants among those studied. No evidence was found for the etiologic role of environmental agents, but more detailed investigations are needed. It is suggested that germ-cell tumors may partly result from hormonal factors activated at menarche and sustained through the child-bearing age.

- 5142 THE INCIDENCE OF MALIGNANT LYMPHOMA AND MULTIPLE MYELOMA IN HIROSHIMA AND NAGASAKI ATOMIC BOMB SURVIVORS, 1945-1965. (E.) Nishiyama, H. (Atomic Bomb Casualty Commission, Hiroshima, Japan), R. E. Anderson, T. Ishimaru, K. Ishida, Y. Ii and N. Okabe. *Cancer* 32(6):1301-1309, 1973.

The incidence of malignant lymphoma and multiple myeloma among survivors of the atomic bomb in Nagasaki and Hiroshima was studied. The data revealed an increased rate of occurrence of malignant lymphoma and multiple myeloma among Hiroshima survivors exposed to 100 rad or greater. This relationship was not evident in the data from the Nagasaki survivors. This discrepancy may have been due to qualitative discrepancies between the two bombs and the resultant radiation spectra and/or biological inconsistencies between the two populations. It was also found that there was an increased prevalence of lymphosarcoma, reticulum cell sarcoma, and Hodgkin's disease among persons exposed to 100 rad or more. In general, the incidence of lymphoma and multiple myeloma was greater among persons less than 25 yr of age at the time of exposure than among those aged 25 yr or more. The diseases also developed more quickly in the younger people than in the older people and in persons exposed to 100 rad or more as opposed to less than 100 rad. With one exception, there were no apparent radiation-dose-related differences with respect to the clinical evolution of the diseases or the attendant laboratory abnormalities.

- 5143 THE EPIDEMIOLOGIC PATHOLOGY OF OVARIAN CANCER. (E.) Berg, J. W. (U. Iowa Coll. Med., Iowa City) and S. M. Baylor. *Human Pathol* 4(4):537-547, 1973.

Data from two epidemiological studies of ovarian cancer, each covering over 10,000 cases divided by histologic type, were analyzed. One study covered international incidence rates, while the other dealt with incidence rates within the United States. The international data indicated that all main types of ovarian cancer in middle aged or older women seem to have parallel geographic distributions. The U.S. data indicated that most ovarian cancers, especially malignant teratomas, occur more frequently among blacks. With regard to age, most dysgerminomas occurred in girls and younger women, malignant teratomas showed a bimodal age distribution, adenoacanthomas

occurred primarily in women between 40 and 59 years of age, adenocarcinomas and carcinomas occurred in older patients, and most of the rest occurred in women 30 yr of age or older. Survival figures corresponded closely to the degree of localization of the cancer, with the survival time varying with the particular type of cancer. The mortality rate among blacks with mucinous carcinomas was twice that found among whites with the same type of cancer. The best relative survival rates were found among young adults; cancers occurring late in life appear to be more malignant. The rare ovarian cancers are discussed briefly. In addition to specific diagnoses, ovarian cancers carry nonspecific diagnoses; these are discussed in terms of how they are used and how cancers so diagnosed relate to cancers with more specific diagnoses. Because of nonspecific diagnoses, clinicians are being denied important information.

5144 INFLUENCE OF PARABIOSIS ON TUMOUR GROWTH IN TUMOUR BEARING ANIMALS. (E.)

Postuma, H. S. (Lab. Exp. Surg., U. Amsterdam, The Netherlands). *Arch Chir Neerl* 15(3):267-282, 1973.

The effect of parabiosis, or cross-circulation, on the growth of a transplanted mammary carcinoma originally induced in the C57 black strain by mammary tumor virus was studied in 150 experiments on highly inbred female F₁ hybrid mice crossing C57 black and CBA strains. Tumor growth was significantly retarded if cross-circulation was made 3 days before grafting or 3 and 6 days after grafting. Parabiosis 9 days after grafting had no retarding effect. When parabiosis was made between tumor bearing animals and immune animals obtained by separation of the anastomosis 14 days after parabiosis with a tumor bearing animal, a significant influence on tumor growth was observed. Immunodiffusion and immunofluorescence showed the presence of humoral tumor specific antibodies in the tumor-host-system. It is concluded that parabiosis is a valuable technique for demonstrating and studying the specific immunological anti-tumor activity of the host after actively growing tumors have been implanted.

5145 CORRELATION BETWEEN SOME MEASURES AS INDEX OF MAMMARY TUMOR SIZE. (E.) Nagasawa, H. (Nat'l. Cancer Ctr. Res. Inst., Tokyo, Japan) and R. Yanai. *Gann* 64(4):405-406, 1973.

Correlation coefficients were calculated between every two of the following six measures which are commonly employed as the index of tumor size using spontaneous mammary tumors of mice as the material: arithmetical or geometrical mean of the two major diameters, wet or dry weight, total DNA, and DNA content/mg dry wt. Spontaneous mammary tumors of several sizes were obtained from virgin and multiparous Swiss albino mice, C3H/He, C3H/HeMs, and C3HeB/FeJax. The measures examined were highly correlated with each other in any strain ($P < 0.001$), except for DNA/mg dry weight. The results suggest the validity of applying these measures of tumor size even to necrotic mammary tumors.

5146 CANCER OF BLADDER. EXPERIENCES OF A HEALTH MAINTENANCE ORGANIZATION. (E.) Campbell, H. J. (Kaiser Fdn. Hosp., Los Angeles, Ca.). *Urology* 2(6):637-642, 1973.

Records of the tumor registries of the Seven Southern California Kaiser Foundation Health Plan hospitals were reviewed and data obtained were compared with data from the California Tumor Registry and the Barnes Hospital (St. Louis, Missouri) Tumor Registry. The Kaiser Health Plan is a pre-paid, comprehensive medical care program. The annual age-adjusted incidence rate was slightly higher among the Kaiser Health Plan members than the populace of Alameda County, California, although rates for both groups have decreased during the latter years. Median age at the time of diagnosis was 5 yr younger for the Kaiser patients (62 yr), than for California (67.6 yr). Although the annual age-adjusted incidence rate for men in the Kaiser Health Plan had declined in recent years, there has been a co-incident slight rise in the rate among women. The male:female ratio of patients with cancer of the bladder was almost twice as high in the Kaiser membership (4.3 to 1) as in the rest of the State of California and Alameda County, California (2.5 to 1). The percentage of Kaiser Health Plan members with bladder cancer who had recurrences during the first yr after diagnosis rose from 34 to 41% during the study period. Median delay time from onset of symptoms to date of diagnosis was similar among the Kaiser Health Plan and California tumor Registry patients, being approximately three months for the more common histologic types of bladder cancer. The impracticality of screening patients to diagnose cancer of bladder earlier is discussed.

5147 REPORT OF THE ADVISORY COMMITTEE ON ASBESTOS CANCERS TO THE DIRECTOR OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER. (E.) Gilson, J. C. (Llandough Hosp., Penarth, Glamorgan, Wales). *Br J Ind Med* 30(2):180-186, 1973.

In October 1972 the International Agency for Research on Cancer (IARC) held an international conference at Lyon, France, to review evidence relating asbestos to cancer. At the final session the Advisory Committee on Asbestos Cancers presented its report to the Director of the IARC. The report is divided into two sections, the first of which reviews present evidence linking exposure to asbestos dust to cancers, especially the evidence obtained since the 1964 meeting of the International Union Against Cancer Working Group on Asbestos Cancers. The second part contains recommendations for further research in the areas of epidemiology, pathology, and physics and chemistry. Priorities for work of immediate and long-term value are indicated. Epidemiologic research must be directed toward simultaneous study of more than one type of cancer. High priorities for pathology projects are methods for determining the amounts, types, and structural features of asbestos in tissue; investigation of the effects of reduction of asbestos exposure to levels below those producing asbestosis; and monitoring by immunologic methods of populations exposed to asbestos. Of im-

portance for physical and chemical studies are the effects of fiber size and shape on pulmonary retention, the site of deposition, the migration of fibers within the body, and their carcinogenic or other biological activity.

5148 GERMINAL CELL TUMORS OF THE TESTIS - INCIDENCE AND RACIAL PREDILECTION. (E.)

Faller, W. (10th Med. Lab., U. S. Army Med. Corps, APO 09180). *Med Bull US Army Eur* 30(11):301-303, 1973.

The incidence and racial distribution of germinal cell tumors of the testis as reported to the 10th Medical Laboratory over the past 5 yr is examined. During this period 43 cases were reported out of the approximately 200,000 men stationed in Europe or an incidence of 4.3/yr/1.0x10⁵ men. This indicates an increase in incidence of 1.4 cases/1.0x10⁵ men since the period 1940-1947. In 40 of 43 cases the clinical history included the race of the man from whom the tissue was taken; not one of the 40 was a Negro. The distribution of tumor type in these 43 cases is as follows: seminoma, 11 cases (25.6%); teratoma, pure or with seminoma, 2 cases (4.6%); teratoma with either embryonal carcinoma or choriocarcinoma or both and with or without seminoma, 21 (48.8%); embryonal carcinoma, pure or with seminoma, 8 (18.6%); choriocarcinoma pure or with either embryonal carcinoma or seminoma or both, 1 (2.3%).

5149 NASOPHARYNX CANCER IN WEST MALAYSIA-INCIDENCE 1963-1965. (E.) Dharmalingam, S. K. (Gen. Hosp., Kuala Lumpur, Malaysia) and S. H. Wong. *Australas Radiol* 17(3):261-265, 1973.

A retrospective analysis of the experience of the Radiotherapy Unit in General Hospital in Kuala Lumpur shows a high incidence of cancer of the nasopharynx among the Chinese in Malaysia and a medium incidence among the Malays. There does not appear to be an increased frequency among the Indians. There is some evidence that the disease is of lesser frequency than among the Chinese residents of Hong Kong which may be partly due to the reduction in the intensity of carcinogenic stimulation.

5150 QUANTITATIVE CYTOLOGY AND CYTOCHEMISTRY OF HODGKIN'S TISSUE LABELLED *IN VIVO* WITH TRITIATED THYMIDINE. (E.) Peckham, M. J. (Royal Marsden Hosp., London, England). *Br J Cancer* 28(4):332-339, 1973.

Morphological properties of the lymph node and spleen cells from two patients with Hodgkin's disease were studied; the labelling pattern with ³H-thymidine was studied in *in vivo* labelled imprints and histological sections. The cellular compositions of two lymph nodes removed simultaneously from a patient with mixed cellularity Hodgkin's disease were remarkably similar despite a marked discrepancy in nodal volume. This indicates that the increase in tumor volume in Hodgkin's disease reflects the expansion of the entire cellular

population of the node, so that it maintains its relative proportionality. In a second patient with lymphocyte predominance/mixed cellularity Hodgkin's disease, histological progression was associated with an increase in aneuploidy of the Hodgkin cell line and with an apparent increase in the large basophilic blast cells but not the Hodgkin cells. The splenic Hodgkin's disease showed less aneuploidy. These data are consistent with the hypothesis that the Hodgkin's disease process constitutes an abnormality of lymphocytes.

5151 POPULATION SCREENING FOR CERVICAL CARCINOMA IN FREDERIKSBURG BOROUGH: RESULTS OF SECOND AND THIRD RESCREENINGS, 1966-1972. (E.) Gad, C. (Frederiksberg Hosp., Copenhagen, Denmark) and F. Koch. *Dan Med Bull* 20(5):141-143, 1973.

The results of the second and third rescreenings for cervical carcinoma in the borough of Frederiksberg, Copenhagen, Denmark, are presented. The irrigation smear method (Cytospinette) was used. The participation rates were 84.8% (of 8,538) and 78% (of 8,190), resp. The rates/1000 women were: for carcinoma *in situ* 1.1 and 0.3, for invasive carcinoma 0.7 and 0.0. The results of repeated cytological screenings indicate that, after a few rescreenings, very few further invasive carcinomas will be found, and rates of carcinoma *in situ* will also decrease. These rates are in agreement with those of other screening programs. There is good evidence that cytological mass screening of this type will lead to a relative increase in detection of early stages of cervical carcinoma and a decrease in the incidence of invasive carcinoma and mortality.

5152 INFECTIOUS MONONUCLEOSIS IN DENMARK. EPIDEMIOLOGICAL OBSERVATIONS BASED ON POSITIVE PAUL-BUNNELL REACTIONS FROM 1940 TO 1969. (E.) Rosdahl, N. (Inst. Med. Microbiol., U. Copenhagen, Denmark), S. O. Larsen and A. B. Thadstrup. *Scand J Infect Dis* 5(3):163-170, 1973.

An epidemiological investigation was made in Denmark covering data from 17,073 patients in whose serum were found heterophile antibodies by a positive Paul-Bunnell reaction at Statens Seruminstitut in Copenhagen during 1940-1969. The purpose of this study was to aid in another investigation to determine if persons having a history of infectious mononucleosis (IM) are more likely to suffer from Hodgkin's disease, leukemia, or some other diseases at some later date. The general incidence of IM was noted to have increased through the time period studied; a steep unexplained increase occurred in 1960. Serological confirmation by the Paul-Bunnell reaction were far more frequent in metropolitan than in rural areas. The relation between Paul-Bunnell verified cases and the total number of notified cases depended on age. The rate of serological confirmations was highest between 15-44 yr of age. A high incidence occurred just below the age of 20 yr. The sex distribution was about equal; however, the peak age for females was about 2 yr lower than in males. During the study, the peak age of inci-

dence dropped by 2 yr for each sex. The most marked decrease in incidence was in children below the age of 9 yr. No significant seasonal differences were noted, except in school children who had low disease incidence in the summer months and peak incidence for the period October to January, with 39.3% of all cases occurring in these months.

- 5153 OESOPHAGEAL CANCER STUDIES IN THE CASPIAN LITTORAL OF IRAN: THE CASPIAN CANCER REGISTRY. (E.) Mahboubi, E. (Inst. Public Hlth Res., U. Teheran, Iran), J. Kmet, P. J. Cook, N. E. Day, P. Ghadirian and S. Salmasizadeh. *Br J Cancer* 28(3):197-214, 1973.

The results of the first 3 yr of cancer registration on the Caspian Littoral are described. The main finding, confirming previous reports, is a very large variation with the region of the incidence of esophageal cancer. Possible sources of bias are considered and shown to contribute little to the pattern of incidence. There is a thirty-fold variation in the incidence across the regions among women; among men a ten-fold variation. In the north-east of the region the tumor is at least as common in women as in men, and is more common than almost any tumor anywhere in the world. In this region the actuarial risk of developing cancer of the esophagus before age 65 is 1 in 6 for both males and females. The regional pattern of variation for esophageal cancer parallels changes in certain ecological variables. Incidence increases with a decline in rainfall and with the associated changes in soil types, natural vegetation and farming practice. Among other tumors, stomach cancer has a strikingly uniform incidence by comparison; breast cancer shows an incidence gradient of opposite slope.

- 5154 RESULTS OF MASS SCREENING FOR CARCINOMA OF THE CERVIX BY 15 PEKING HOSPITALS. (E.) Anonymous (Peking Coordinating Group, Mass Screening of Cervix, People's Republic of China). *Chinese Med J* (9):113, 1972.

- 5155 THE FREQUENCY OF INTRACRANIAL NEOPLASMS IN NEWFOUNDLAND. (E.) Maroun, F. B. (Gen. Hosp., St. John's, Newfoundland, Canada) and J. C. Jacob. *Can J Public Health* 64(1):53-57, 1973

- 5156 ASSESSMENT OF MORTALITY FROM MALIGNANT NEOPLASMS IN THE YEARS 1963-1967. (E.) Gadowska, H. (Inst. Oncol., Warsaw, Poland), T. Kosbarowski and Z. Karewicz. *Sante Publique* 124(4):429-442, 1971.

- 5157 MULTIPLE PRIMARY MALIGNANT TUMORS OF THE RESPIRATORY TRACT IN MAN. (E.) Epstein, S. S. (Case Western Reserve U., Sch. Med., Cleveland, Ohio). *Vth Perugia Quadrennial Intl Conference on Cancer* 35, 1973.

- 5158 ROLE OF KINETIN (6-FURFURYLAMINOPURINE) IN THE STIMULATION OF CELL DIVISION (*Allium cepa* L.). (Rus.) Balasanian, D. S. (Dept. Genetics) Cytol., Erevan State U., USSR). *Biol Zh Armenii* (5):70-74, 1973.

- 5159 ANALYSIS OF MASS SCREENING FOR CERVICAL CANCER IN A TIENTSIN DISTRICT. (E.) Anonymous (1st, 2nd Treatment Hosp., Tientsin, People's Republic of China). *Chinese Med J* (9):114, 1973.

- 5160 DIURNAL RHYTHM OF CELL DIVISION IN TUMORS. (E.) Kharlampovich, S. I. (Res. Inst. Med. Radiology, USSR, Acad. Med. Sci., Obninsk) and T. P. Svinogeeva. *Bull Exp Biol Med* 75(2):158-160, 1973.

- 5161 THE FORMATION OF THYROGLOBULIN IN HUMAN THYROID MEDULLARY CARCINOMA. (E.) Ljunggren, J.-G. (Karolinska Inst., Stockholm, Sweden), T. Löwhagen and B. Hjern. *Acta Endocrinol* 74:105-110, 1973.

- 5162 SMALL CELL CARCINOMA OF THE LUNG. CLINICO-PATHOLOGICAL STUDIES. (E.) Takita, H. (Roswell Park Mem. Inst., Buffalo, N.Y.), A. Brugarolas, P. Marabella and R. G. Vincent. *J Thorac Cardiovasc Surg* 66(3):472-477, 1973.

- 5163 PROLIFERATIVE CAPACITY OF LEUKEMIA CELLS FROM THE HUMAN BONE MARROW: CORRELATION WITH CYTOLOGICAL FEATURES, CLINICAL COURSE AND EFFECTIVENESS OF THERAPY. (Fr.) Faille, A. (St. Louis Hosp., Paris, France), Y. Najean and J. Bernard. *Nouv Presse Med* 2(14):889-894, 1973.

- 5164 PREVALENCE AND MORTALITY FOR BREAST CANCER IN THE ARMENIAN SSR OVER A 30 YEAR PERIOD. (Rus.) Gaiserian, A. M. (Armenian Inst. Radiol. Oncol., Tbilisi, USSR). *Zh Eksp Klin Med* 12(1):108-111, 1972.

- 5165 BLAST CELL PROLIFERATION IN ACUTE LEUKEMIA. (Rus.) Kozinets, G. I. (Ctr. Inst. Hematol. Blood Transfusion, Moscow, USSR), N. A. Gerasimova, S. M. Dul'tsina and L. G. Kovaleva. *Arkh Patol* 35(1):62-66, 1973.

- 5166 SMALL-CELL DYSPLASIA AND *IN SITU* CARCINOMA OF MAMMARY DUCTS AND LOBULES. I. INCIDENCE AND PREDISPOSING CONDITIONS. (E.) Toker, C. (Mt. Sinai Sch. Med., New York, N.Y.). *Mt Sinai J Med* 40(6):780-782, 1973.

5167 MEGALOBLASTOSIS IN HEMATOLOGIC MALIGNANCY.
 (E.) Bart, J. B. (Kelsey Seybold Clin.,
Houston, Texas). *S Med J* 66(9):981-983, 1973.

5168 TUMORS OF THE UPPER URINARY TRACT: REGION-
 AL STATISTICS AND ATTEMPT TO ESTABLISH AN
ANATOMICOPATHOLOGICAL DEFINITION BY MATHEMATICAL
METHODS ON THE BASIS OF 22 CASES. (Fr.) Bittard,
M. (U. Hosp. Ctr., Besancon, France), R. Nataf, G.
Camelot and D. Loiseau. *J Urol Nephrol*
78(12 bis):275-278, 1972.

See also:

* (Rev): 4802

- 5169 HISTOCHEMISTRY AND FINE STRUCTURE OF BRONCHIAL CARCINOID TUMOURS. (E.) Hage, E. (Odense Hospital, Denmark). *Virchows Arch (Pathol Anat)* 361(2):121-128, 1973.

The histological, histochemical and ultrastructural features of six carcinoid tumors of the larger bronchi were investigated. Electron microscopy and methods known to stain endocrine cell granules selectively allowed differentiation of three types of endocrine cells. Two of these cell types were similar to endocrine cells normally found in the pulmonary epithelium of the human fetus. These cells had small, round membrane-bound secretory granules of uniform size and shape, or much larger, round secretory granules tightly surrounded by a membrane and almost homogeneous in appearance. The third cell type was characterized by large polymorphic secretory granules, vesiculated or tightly surrounded by a membrane. These cells were reactive to staining with the argentaffin silver method and were quite similar to the enterochromaffin cell known from the digestive tract. Scattered mastocytes which reacted to some of the granule staining methods were easily identified by electron microscopy.

- 5170 NEUROBLASTOMA CELL DIFFERENTIATION: A TISSUE CULTURE STUDY USING TIME-LAPSE CINEMATOGRAPHY. (E.) Booher, J. (Pasadena Fdn. Med. Res., California), M. Sensenbrenner and P. Mandel. *Neurobiology* 3(5):335-338, 1973.

Continuous records were made on two clonal lines of mouse neuroblastoma cultures during the evoked phases of their differentiation cycle. The taking rate of the time-lapse sequences was one frame/min which, when projected, enhanced the time span 1440 times. Atypical neuronal fiber process formation occurred. The process formation and retraction was achieved by the migration of the cell body rather than by the more typical fiber outgrowth and extension. The observation in these studies that the normal morphological formation of neuronal processes as seen in the dissociated central nervous system and "p.n.s." cultures is not stimulated in neuroblastoma cells, makes the use of these cells questionable as a test object for morphological neuronal differentiation.

- 5171 INTERACTING CELL POPULATIONS AFFECTING GRANULPOIETIC COLONY FORMATION BY NORMAL AND LEUKEMIC HUMAN MARROW CELLS. (E.) Messner, H. A. (Inst. Med. Sci., U. Toronto, Canada), J. E. Till and E. A. McCulloch. *Blood* 42(5):701-710, 1973.

Marrow from 28 nonleukemic individuals was separated by adherence to glass or plastic into nonadherent (NA) and adherent populations. The NA populations were more dependent for colony formation in culture on added colony-stimulating activity (CSA) than unseparated marrow suspensions, thereby providing an improved assay for CSA and CSA-producing cells. Heavily irradiated marrow cells

which had lost their ability to form colonies were added to NA cells; there was a linear relationship between the number of added irradiated marrow cells and colony formation. In another experiment, colony formation among NA cells increased as a linear function of the number of adherent cells added, while in another experiment homologous irradiated peripheral blood cells were used to induce colony formation among NA cells. Assays for CSA producing cells were made on the marrow of four patients with acute leukemia. In all four cases, leukemic marrows were less effective than normal marrows in promoting colony formation by NA cells of normal individuals. A mixture of irradiated unseparated leukemia marrow and NA cells yielded no colonies. These data support the contention that normal human marrow contains both granulopoietic colony-forming cells (CFU-C) and cells capable of producing molecules required by these progenitors for proliferation and differentiation in culture. The CSA-producing cells of the leukemic patients studied were abnormal either in number or productive capacity.

- 5172 GIANT LYSOSOME-LIKE STRUCTURES IN PROMYELOCYTIC LEUKEMIA. ULTRASTRUCTURAL AND CYTOCHEMICAL OBSERVATIONS. (E.) Mintz, U. (Beilinson Hosp., Tel-Aviv, Israel), M. Djaldetti, L. Rozenszajn, J. Pinkhas and A. de Vries. *Biomedicine* 19(10):426-430, 1973.

The promyelocytes of a 25-yr-old woman with acute promyelocytic leukemia and disseminated intravascular coagulation were studied using electron microscopy and cytochemical techniques. The outstanding features in the promyelocytes from this patient were giant granules and Auer bodies. The giant granules were lysosomal in nature and differed both biochemically and structurally from normal cytoplasmic granules. The Auer bodies also appeared to be lysosomal in nature. It is suggested that these two structures may have a common origin and that they may be related to the intravascular coagulation in this patient.

- 5173 MAMMARY NODULIGENESIS AND TUMORIGENESIS IN PATHOGEN-FREE C3Hf MICE. (E.) Medina, D. (Baylor Coll. Med., Houston, Tex.), J. Vaage and R. Sedlacek. *J Natl Cancer Inst* 51(3):961-965, 1973.

Mammary noduligenesis and tumorigenesis were studied in pathogen-free C3Hf/Sd female mice. The mice experienced a normal breeding regimen for up to five pregnancies, then were retired to live out their lifespan. Of 38 mice, 29 died of mammary tumors with a mean latent period of 700 days, 4 died of other neoplasms, and 5 died of non-neoplastic causes. It seemed that mice kept free from accidents or infectious diseases would ultimately develop neoplasms. The rate of mammary tumorigenesis in pathogen-free mice paralleled the rate of tumorigenesis in conventional mice. The high mammary tumor incidence reflected the fact that the whole population of mice survived long enough to develop such tumors. Thus,

the low oncogenic potential noted for the nodule-inducing virus was related to the late age of onset of mammary tumors in C3Hf mice, rather than to the ultimate tumor incidence.

- 5174 EFFECT OF ADENOSINE 3',5'-MONOPHOSPHATE ON THYMIDINE KINASE IN TUMOR CELLS. (E.) Hamazaki, T. (Chiba U. Sch. Med., Japan). *Gann* 64(3):219-226, 1973.

Thymidine kinase was isolated from Yoshida ascites sarcoma cells and partially purified by DEAE-cellulose column chromatography. Its elution profile showed two distinct peaks, with the peak I enzyme having a molecular weight of about 140,000. On treatment of the peak II enzyme with RNase, new peaks of activity with molecular weights of 200,000, 130,000, and 75,000, appeared. These results indicate that the peak II enzyme may have a complex form and contain RNA. The peak I enzyme was inhibited by cyclic adenosine 3',5'-monophosphate (AMP) and was found in tumor cells as well as in regenerating liver. The peak II enzyme was stimulated by the addition of cyclic AMP and was found only in tumor cells; when this enzyme was incubated with cyclic AMP, it seemed to cleave into five portions. The overall activity of thymidine kinase in tumor cells does not appear to be affected by the addition of cyclic AMP, indicating that cyclic AMP is not important as a regulator of thymidine kinase in tumor cells.

- 5175 ISOLATION OF SOLUBLE ISOTYROSINASES EXTRACTED FROM A MELANOMA IN A HORSE. (Fr.) Kleisbauer, J. P. (Unit 119, INSERM, Marseille, France), G. Profisi-Centa and R. Roubin. *C R Soc Biol* 167(1):87-89, 1973.

Melanomas from gray horses were homogenized in 0.9% NaCl and centrifuged. Electrophoresis of the supernatant on 5% polyacrylamide gel revealed three tyrosinase isoenzymes (T_1 , T_2 and T_3). T_1 migrated the furthest and had a molecular wt of $65,000 \pm 3500$. The molecular wt of T_2 and T_3 were $51,000 \pm 3000$ and $50,000 \pm 3000$, resp. T_1 accounted for 30% of the total isoenzymes present and, contrary to reports in the literature, was not converted into T_2 and T_3 after 24 hr at 30°C . A fourth isoenzyme was found in some tumors, either between T_1 and T_2 or immediately after T_3 . Proteins in the supernatant were precipitated with 30-70% sodium sulfate, centrifuged, dialyzed against phosphate buffer (pH 7) and filtered through a column of diethylaminoethyl (DEAE) Sephadex A 25 to adsorb melanine and other proteins. The precipitate obtained by salting out contained only T_2 and T_3 after it was eluted from DEAE Sephadex with phosphate buffer. These isoenzymes were separated into two peaks by eluting from DEAE Sephadex with Tris HCl buffer (pH 8.4) with increasing ionic strengths. The molecular wt of these isoenzymes were the same as those found in the supernatant. Study of the chemical properties of these isoenzymes may make it possible to produce specific antisera for use in the treatment of melanomas.

- 5176 INHIBITOR OF PYRIMIDINE METABOLISM FROM TUMOR TISSUES. (E.) Arima, T. (Sch. Med., Tokushima U., Japan) and S. Fujii. *Biochem Biophys Res Commun* 55(2):410-416, 1973.

Two inhibitors of rat liver 5'-nucleotidase and d-uridine monophosphate (dUMP) kinase *in vitro* were isolated from the $105,000 \times g$ supernatant fraction of rapidly proliferating tissues, such as Yoshida sarcoma. Inhibitory activity was abolished by heating in boiling water for three min. Two peaks of inhibitor activity were separated by zone electrophoresis of the supernatant fraction. One peak, migrating to the cathode, inhibited both enzymes, while the other peak, which migrated to the anode, inhibited only dUMP kinase. The latter inhibitor did not adsorb to a DEAE-cellulose column. The molecular wt of the two inhibitors were estimated at 500,000 and 50,000 based on their elution pattern from Sephadex G-200 columns. The inhibitors were not found in normal rat liver but could be found in rapidly regenerating liver and Ehrlich ascites cells. The presence of such inhibitors presumably accounts for the reduced activity of 5'-nucleotidase and dUMP kinase seen in rapidly proliferating tissues.

- 5177 PULMONARY ADENOMATOSIS IN AGEING MICE. (E.) Baillif, R. N. (Tulane U. Sch. Med., New Orleans, La.) and E. L. Jones. *J Comp Pathol* 83(4):597-603, 1973.

The clinical and histological features of pulmonary adenomatosis were studied in female CD-1 mice inoculated i.p. with live Ehrlich ascites tumor (EAT) cells or explanted s.c. or i.p. with fragments from various spontaneous murine tumors. Untreated mice were maintained as controls. Of 482 treated mice, 101 developed pulmonary adenomatosis which process was primarily carcinomatous. Typical pulmonary adenomatosis nodules were composed of a core of darkly staining adenomatous tubules or cords sometimes modified into complex papillations; the tubular area was surrounded or intermixed with lightly staining cell clusters with or without lumens. The tubules and cords appeared to arise from the bronchiolar lining, whereas the cell clusters apparently arose from the alveolar lining. The histological pattern became highly variable as the neoplastic process progressed. Metastases in organs other than the lung itself were found in only seven mice. The frequency of pulmonary adenomatosis was significantly higher than that of controls (16.9%) only for animals explanted with murine tumor fragments (30.8%, $P < 0.05$). The incidence in EAT-inoculated animals was 17.5%.

- 5178 CONTROL OF PLASMA ALDOSTERONE IN PRIMARY ALDOSTERONISM: DISTINCTION BETWEEN ADENOMA AND HYPERPLASIA. (E.) Ganguly, A. (Stanford U. Sch. Med., California), G. A. Melada, J. A. Luetscher and A. J. Dowdy. *J Clin Endocrinol Metab* 37(5):765-775, 1973.

Plasma aldosterone fell during the morning in 9 of 11 ambulatory patients with hyperaldosteronism due to aldosterone-producing adenoma. However,

in 7 cases of idiopathic adrenal hyperplasia, standing increased plasma aldosterone. The diurnal curve of plasma aldosterone in aldosterone-producing adenoma declined from early morning to late evening, parallel with falling plasma cortisol, in spite of stimulation of renin by posture, sodium depletion or spironolactone. Plasma aldosterone was increased after ACTH injection and decreased after dexamethasone in aldosterone-producing adenoma. The ordinary diurnal rhythm of plasma aldosterone does not parallel plasma cortisol and is not consistently altered by dexamethasone in idiopathic adrenal hyperplasia. However, aldosterone secretion can be stimulated by ACTH. Plasma aldosterone in idiopathic adrenal hyperplasia is consistently increased by standing and by a low-sodium diet. Plasma renin concentration and plasma aldosterone are increased after spironolactone much more so than in cases of aldosterone-producing adenoma. It is concluded that plasma aldosterone in idiopathic adrenal hyperplasia responds to posture-related stimuli to a greater degree than aldosterone-producing adenoma. The differences in diurnal rhythm and physiological control of plasma aldosterone can be used in differential diagnosis of the two disorders.

- 5179 BIOCHEMISTRY OF BREAST CYST FLUID. (E.) Fleisher, M. (Memorial Sloan-Kettering Cancer Ctr., New York, N.Y.), G. F. Robbins, C. N. Breed, Jr., A. A. Fracchia, J. A. Urban and M. K. Schwartz. *Clin Bull Sloan-Kettering Cancer Ctr* 3(3):94-97, 1973.

Breast cyst fluid from 43 patients was analyzed biochemically for steroid, enzyme, protein, and inorganic ion content. The chemical composition was found to be highly variable and quite different from that of other body fluids. 17,21-Dihydroxysteroid content ranged from 0 to 7.0 $\mu\text{g/ml}$, and 17-ketosteroids from 1.0 to 43.5 $\mu\text{g/ml}$. No apparent relation was found between cyst fluid calcium and phosphorous levels, and potassium levels were similar to intracellular concentrations suggesting pre-existing tissue breakdown. Total protein levels were very low and albumin was very low to absent. Electrophoretic analysis showed the major protein fractions to be serum α_1 , α_2 and β globulins. Alkaline phosphatase activity was variable, ranging from 0 to 350 IU. Lactic dehydrogenase activity was usually low, indicating the existence of very little RBC breakdown. Fluid from several cysts showed high levels of carcinoembryonic antigen, the significance of which is unknown.

- 5180 DIFFERENT PROPERTIES OF MICROSOMAL UDP-GLUCURONYLTRANSFERASE IN BUFFALO RAT LIVER AND A CLONAL STRAIN OF RAT HEPATOMA CELLS DERIVED FROM THE SAME RAT STRAIN. (E.) Winsnes, A. (U. Hosp., Oslo, Norway) and H. E. Rugstad. *Acta Pharmacol Toxicol* 33(3):161-176, 1973.

Optimal conditions were established for the synthesis of o-aminophenol and p-nitrophenol glucuronides by a clonal strain of rat hepatoma cells (MH₁C₁) in culture. Properties of glucuronyltransferase were

studied in homogenates of cultured hepatoma cells, s. c. tumors derived from these cells in rats, as well as livers of Buffalo rats. In rat liver 83-90% and 92-95% of the glucuronyltransferase activity in homogenates were latent to p-nitrophenol and o-aminophenol, resp.; this was most pronounced in male rats. Using homogenates of cultured hepatoma cells, digitonin activated 1.3 and 1.8-fold only with p-nitrophenol and o-aminophenol as acceptors, resp. Triton X-100, UDP-N-acetylglucosamine and diethyl-nitrosamine did not increase glucuronyltransferase activity. In homogenates of hepatoma tumors derived from the same cells injected s.c. into Buffalo rats, a 1.5- to 2-fold higher degree of activation was found. The rate of p-nitrophenol glucuronide synthesis by cultures of hepatoma cells increased up to a concentration of 0.10 mM of the aglycone in the growth medium. The maximal rate of o-aminophenol glucuronidation in hepatoma cell cultures corresponded to that for homogenates with 0.25 - 0.50 mM UDP-glucuronate added to the incubation mixture. This value agrees with the assumed intracellular levels of UDP-glucuronate in the liver cell *in vivo*.

- 5181 ROLE OF DIFFUSION BOUNDARY LAYER IN CONTACT INHIBITION OF GROWTH. (E.) Stoker, M. G. P. (Imperial Cancer Res. Fund Lab., London, England). *Nature* 246(5430):201-203, 1973.

The role of a diffusion boundary layer in contact inhibition of growth was studied in confluent 3T3 cell cultures exposed to a stream of medium pumped at an increased velocity. Wounds were introduced into such cultures by scraping with a razor blade, and the stream of medium was directed across the edge of the wound toward the cell sheet. Both mitotic index and the rate of ³H-thymidine incorporation as determined by autoradiography were increased five- and eight-fold, resp., in the area of greatest velocity as compared with other areas along the wound edge. Cinematography revealed no loss of cells but did reveal increased local cell movement in the area of the stream. Microdensitometer tracings showed that the uptake of dilute Giemsa stain was also enhanced in cells within the band of increased fluid velocity. These results indicate that concentration changes in the microenvironment could account for the highly localized growth of 3T3 cells at the edge of wounds and presumably also at the circumferences of cell colonies, thus offering an alternative to alterations in cell-cell contact as a mechanism.

- 5182 ELECTRON MICROSCOPIC STUDIES OF HUMAN PITUITARY TUMORS. I. CHROMOPHOBIC ADENOMAS. (E.) Schechter, J. (U. Southern California, Sch. Med., Los Angeles). *Am J Anat* 138(3):371-385, 1973.

Electron microscopy was used to study 10 cases of chromophobic adenoma of which 7 were associated with hypopituitarism and 3 with acromegaly. Morphological variations in parenchymal cell types were slight among the tumors and a single cell

type predominated and was common to both varieties of the tumor. Varying amounts of secretory granules measuring 80-200 m μ or more in diameter were found in the predominant cell type which also had an electron-opaque core and perigranular halo. The core material was homogeneously electron-opaque in some granules and particulate in others. Many secretory granules also contained aggregates of clear vesicles. Lysosomes were abundant and a consistent feature of the tumor cells. Thus cell features are significantly altered in the cells of both varieties of chromophobic adenoma so that ultrastructural criteria used to identify cell types in normal pituitary glands are not applicable to the tumor cells. Chromophobic adenomas with acromegaly varied more in the features of the predominant cell type than did chromophobic adenomas with hypopituitarism. Marked variations in the number and structure of mitochondria from cell to cell was common to both varieties. Mitochondria occasionally filled the cytoplasm.

- 5183 ROLE OF SUN EXPOSURE IN THE ETIOLOGY OF MALIGNANT MELANOMA: EPIDEMIOLOGIC INFERENCE. (E.) Movshovitz, M. (Chaim Sheba Med. Ctr., Tel Hashomer, Israel) and B. Modan. *J Natl Cancer Inst* 51(3):777-779, 1973.

A nationwide study of malignant melanoma in Israel (1961-67) revealed a high incidence rate in the native-born of European extraction. Rates were intermediate in the veteran foreign-born Europeans, lower in more recent European-born immigrants, and lowest among the Asian- and African-born. A 1:1.5 male-to-female ratio could be accounted for by the relatively higher incidence of lesions of the upper and lower extremities in females. These results indicate a causative role of sun exposure in the etiology of malignant melanoma and demonstrate that the disease incidence is modified by environmental factors.

- 5184 AN ERROR CASCADE MECHANISM FOR TUMOR PROGRESSION. (E.) Wheldon, T. E. (Western Reg. Hosp. Board, Glasgow, Scotland) and J. Kirk. *J Theor Biol* 42(1):107-111, 1973.

According to the "error cascade" theory the fidelity of protein synthesis in a somatic cell deteriorates with age. Many of the direct results of inaccurate protein synthesis will be transient but a minority of these effects will be heritable. If the error cascade develops randomly, a small probability exists that heritable damage to homeostatic molecules, that is those molecules involved in the regulation of mitotic activity, precedes that to any group of essential molecules. In such a case, a viable clone of cells with relative autonomy would develop. Such a clone would exhibit low fidelity of protein synthesis, so providing a rapidly varying population from which the most autonomous members would be selected. Predictions of such a theory include: 1) an association between cancer and aging; 2) a high rate of cell death in tumors; 3) heterogeneity of protein synthesis within a tumor population; 4) oncogenicity

and progression-enhancing capacity of substances causing inaccurate protein synthesis; and 5) deterioration in hormone dependence of tumors.

- 5185 CHOLESTEROL IN CEREBROSPINAL FLUID OF BRAIN TUMOR PATIENTS. (E.) Fleisher, J. H. (Arizona Med. Ctr., Tucson), L. J. Marton, N. R. Bachur and R. S. Mann-Kaplan. *Life Sci* 13(11):1517-1526, 1973.

A sensitive method for assay of total cholesterol (free plus esterified) in one ml of cerebrospinal fluid (CSF) based upon gas-liquid chromatography has been developed. CSF was obtained from one group of 11 patients with brain tumors and a second group of 11 with other neoplasms. The average cholesterol concentration (12.66 μ g/ml) in the CSF of patients with brain tumors (seven with glioblastomas and four with astrocytomas) was significantly higher than the average (3.30 μ g/ml) found in the CSF from patients with neoplasms outside of the central nervous system. The data suggest that assay of total cholesterol in serial samples of CSF obtained during follow-up of brain tumor patients undergoing chemotherapy after brain surgery may serve as a biochemical marker of brain neoplasia.

- 5186 STEROID METABOLISM BY AN OESTROGEN-DEPENDENT INTERSTITIAL-CELL TUMOUR OF A RAT TESTIS. (E.) Jull, J. W. (Cancer Res. Ctr., U. British Columbia, Vancouver, Canada), D. McClellan and S. Coupey. *J Endocrinol* 59(1):7-16, 1973.

Steroid metabolism by a unique, transplantable, estrogen-induced, hormone-dependent interstitial-cell tumor of a rat testis was investigated. After incubation of the minced tumor in medium without substrate 3 α -androstenediol, 3-epiandrosterone and androsterone were detected. There was inconclusive evidence for the presence of smaller amounts of 3 β -androstenediol; no evidence for testosterone. The metabolic products formed by the tumor from [¹⁴C]pregnenolone substrate reflect potential enzymic activities not necessarily connected with steroid synthesis from the intracellular precursors. [¹⁴C]Pregnenolone substrate metabolized after 2 hr to androstenedione, androsterone and a small amount of testosterone. Epi- and dehydroepiandrosterone were probably formed from [¹⁴C]pregnenolone, but were incompletely characterized. This demonstration of 3 α -androstenediol synthesis accounts for the high androgenic activity of the interstitial-cell tumor *in vivo* but this compound alone cannot cause the nature or degree of the associated biological changes.

- 5187 LEUKEMIA IN TWINS (E.) Falletta, J. M. (Baylor Coll. Med., Houston, Texas), K. A. Starling and D. J. Fernbach. *Pediatrics* 52(6):846-849, 1973.

Acute childhood leukemia carries an extraordinary risk of twin concordance - 1:5 for monozygotic twins and

1:8 for dizygotic twins as compared to 1:500 for ordinary siblings. A case history of leukemia in twins is presented in which one twin became ill at nearly six yr of age and the other twin more than six yr later. This case does not follow the general pattern of the disease in twins who normally have the onset of illness within weeks or months of each other. The twins discussed here probably remained at risk of concordant disease because of postnatal factors affecting predisposed siblings. Whether the predisposition was due to genetic or to prenatal environmental factors is not known.

- 5188 THE CYTOPLASMIC RIBONUCLEOPROTEIN PARTICLES OF RAT SARCOMA CELLS. I. THE PARTICLES OF RIBOSOMAL POPULATION AND THE TURNOVER OF THEIR PROTEIN MOIETY. (E.) Votrin, I. I. (First Moscow Med. Inst., USSR), S. S. Debov, S. S. Schischkin and P. Z. Khasigov. *Mol Cell Biochem* 2(1):15-22, 1973.

The metabolism of cytoplasmic ribosomal RNA-particles from rat sarcoma cells was studied after male albino rats were injected with ^{14}C -leucine (200 μCi , i.p.) alone or with $\text{H}_3\text{P}^{32}\text{O}_4$ (200 μCi). The change of the specific activity of the ribosomes with respect to the time after injection of ^{14}C -leucine showed the half-life of ribosomes to be 35 hr. To study the metabolic activity of ribosomes not active in protein synthesis, the sucrose gradient centrifugation method was used to separate them from polyribosomes. Free ribosomal subunits and monoribosomes were isolated 4, 7, and 12 hr after isotope injection and their radioactivity was then determined. The label was found in the ribosomal subunits at the beginning and later on in the monoribosomal fraction. The incorporation of $\text{H}_3\text{P}^{32}\text{O}_4$ into ribosomal subunits occurred at a much greater rate compared to the incorporation of ^{14}C -leucine. These results indicate the existence of a pool of ribosomal proteins in sarcoma cells which may be important in the synthesis of free subunits in sarcoma cells. Such a pool may be characteristic of tumor cells and of growing cells in general.

- 5189 "COELIAC DISEASE AND MALIGNANCY". (E.) Barry, R. E. (Dept. Med., U. Bristol, England). *Tijdschr Gastroenterol* 16(1):23-24, 1973.

Eighteen cases of subtotal villous atrophy who presented with malabsorption were studied with a view to establishing the mechanism by which the mucosa changes are produced. The patients were put on a gluten-free diet, to which ten responded well. The other group of eight patients, however, showed a decreased epithelial cell loss rate associated with a thinning of the mucosa. Two of these patients were found to have lymphosarcoma of multifocal origin arising in the small bowel. Two patients who failed to respond to a gluten-free diet responded well to treatment with prednisone but both patients subsequently relapsed and were found to have primary reticulosarcoma of the small bowel. It is suggested that the mechanism which produces the mucosal abnormality in the gluten non-respon-

sive group is different from that of uncomplicated celiac disease. It appears that a consideration of mucosal dynamics in patients presenting with subtotal villous atrophy may be a useful pointer to the presence of, or a tendency to develop, grave complications, particularly malignant lymphoma.

- 5190 THE ULTRASTRUCTURE OF "PINEALOMA" (SEMINOMA-LIKE TUMOR OF THE PINEAL REGION). (E.) Cravioto, H. (New York U. Med. Ctr., N.Y.) and D. Dart. *J Neuropathol Exp Neurol* 32(4):552-565, 1973.

The ultrastructure of a seminoma-like tumor of the pineal region (atypical teratoma of the pineal) was examined. The tumor was composed of large clear polygonal cells and small lymphocyte-like cells. The origin of the large cells was not clearly established. Although some ultrastructural similarities were found between pineal parenchymal cells and tumor cells, the similarities between germinal and seminoma cells were even more striking. Most of the small cells were identical to lymphocytes; some were leukocytes and some were phagocytes. It is thought that the lymphocytic infiltration represents an immunological response of the host against possible tumor antigen(s). Intact phagocytes were found within the cytoplasm of some of the large tumor cells. This is thought to represent immunologically mediated destruction of the tumor by activated phagocytes. Whether or not the tumor arose in gonadal elements could not be established.

- 5191 LACTIC DEHYDROGENASE ISOZYMES IN BENIGN AND MALIGNANT PROSTATIC TISSUES. (E.) Srinivasan, V. (Division Urology, U. Illinois Hosp., Chicago), E. Keil, R. Villalba, T. Baron and S. S. Clark. *Invest Urol* 11(3):244-247, 1973.

Lactic dehydrogenase (LDH) isoenzyme analysis was carried out in 174 prostatic tissue specimens and the $\text{LDH}_5:\text{LDH}$ ratio (LDH index) was calculated by measuring the peaks in all specimens and by measuring the surface area in 162 specimens. By the former method, only 52% of patients had a positive correlation of LDH index with the pathology. However, when focal carcinoma was eliminated and when surface area was computed, 81% of the carcinoma patients had a positive correlation with the LDH index.

- 5192 ULTRASTRUCTURAL AND BIOCHEMICAL STUDY OF BENIGN GANGLIONEUROMA. (E.) Yokoyama, M. (Fac. Med., U. Tokyo, Japan), K. Okada, A. Tokue and H. Takayasu. *Virchows Arch (Pathol Anat)* 361(3):195-209, 1973.

Ultrastructural and biochemical studies were conducted on three ganglioneuromas to determine if there is a correlation between morphological features and catecholamine synthesis, storage and secretion. Ganglioneuroma tissues showed a striking ultrastructural similarity to sympathetic ganglion cells and neurons, being composed of mature ganglion cells, unmyelinated nerve

bundles, infrequent myelinated nerve bundles and abundant interstitial elements. Distinct Schwann cells were observed with basement membrane surrounding both ganglion cells and nerve processes. Various degrees of morphological differentiation were observed among these tumors, for example, neurofilaments, neurotubules, abundant small clear vesicles (500 Å in size) and large cored vesicles (1000 Å in size). In two of the three tumors studied, catecholamines were detected by chemical assay. Urinary catecholamines were variable. Since this study revealed the presence of large cored vesicles and the existence of catecholamines in ganglioneuroma, it is suggested that the catecholamines are stored in large cored vesicles in the tumor, although unequivocal evidence cannot be provided.

5193 ACUTE LEUKEMIC CELLS. QUALITATIVE AND QUANTITATIVE ELECTRON MICROSCOPY. (E.)

Schumacher, H. R. (Harrisburg Hosp., Pa.), I. E. Szekely, S. A. Park, U. N. M. Rao, D. R. Fisher and S. B. Patel. *Am J Pathol* 73(1):27-46, 1973.

Quantitative and qualitative electron microscopic studies were performed on the leukemic cells of three patients with stem cell leukemia, six patients with acute lymphoblastic leukemia, seven patients with acute monoblastic leukemia, three patients with acute myeloblastic leukemia and seven patients with acute monomyeloblastic leukemia. Significant quantitative differences were noted between some of the leukemic cells in heterochromatin:euchromatin ratios, cell size, granules per cell, amount of endoplasmic reticulum and the number of polyribosomes. Qualitative abnormalities which were found in some cells of all the leukemic groups were observed. These abnormalities included nuclear pockets, deep nuclear indentations (usually not the stem cell), nucleosomes, dilated perinuclear spaces, centrioles located in nuclear pockets (not in the stem cell or lymphoblast), accumulation of microfibrils (greatest in the monoblast and myeloblast), disrupted mitochondria with virus-like particles (none in stem cells), smaller granules and mitochondrial DNA, glycogen, and myelin whorls. The presence of the smaller granules in disrupted mitochondria and the resulting clear areas is probably related to the deranged carbohydrate metabolism of these cells. The presence of virus-like particles within mitochondria may be extremely important, but requires much more investigation.

5194 DEPRESSION OF BONE MARROW DELTA-AMINO-LAEVULIC ACID SYNTHETASE ACTIVITY IN ERYTHROLEUKAEMIA. (E.) Tanaka, M. (Nahoya U. Sch. Med., Japan), T. Hotta and H. Yamada. *Br J Haematol* 25(5):599-606, 1973.

Bone marrow delta-aminolevulinic acid (ALA) synthetase activity and heme synthesis were studied in three patients with erythroleukemia and three controls with refractory normoblastic anemia. ALA synthetase activity and the incorporation of (¹⁴C)glycine and (¹⁴C)ALA into heme were markedly reduced in all three patients with erythroleukemia as compared

with the controls. Following blood transfusions in the erythroleukemic patients, there was some improvement in all three measurements, although all three remained subnormal. These changes tended to correlate with erythroid activity and megaloblastoid changes in the bone marrow. These data are consistent with a primary alteration in the activity or production of ALA synthetase and of additional enzymes in the pathway as associated expressions of the underlying defect in heme biosynthesis in erythroleukemia.

5195 GENERAL INTERSPERSION OF REPETITIVE WITH NON-REPETITIVE SEQUENCE ELEMENTS IN THE DNA OF *XENOPUS*. (E.) Davidson, E. H. (Div. Biol., California Inst. Tech., Pasadena), B. R. Hough, C. S. Amenson and R. J. Britten. *J Mol Biol* 77(1):1-23, 1973.

The arrangement of repetitive and nonrepetitive sequences was studied in the DNA of *Xenopus laevis*. Labeled DNA sheared to various fragment lengths was reassociated to Cot 50 with about 450 nucleotide fragments of unlabeled DNA, and binding of the labeled DNA to hydroxyapatite was measured. Repetitive sequences monitored in this way were present on about 45% of the 450 nucleotide fragments. As DNA fragment length was increased, larger fractions of the DNA were found to contain repetitive elements. Up to 80% of the DNA was found to bind at an average fragment length of 3700 nucleotides. Analysis of the data indicated that a little more than 50% of the genome consisted of closely interspersed repetitive and nonrepetitive sequences. The average length of the repetitive sequence elements was 200 to 400 nucleotides, while the nonrepetitive sequences separating adjacent repetitive sequence elements averaged 600 to 1000 nucleotides. The remainder of the DNA was mainly nonrepetitive, although most of it contained rare interspersed repetitive elements spaced at a minimum of 4000 nucleotides apart. It is concluded that a high degree of order exists in the arrangement of DNA sequences in the *Xenopus* genome.

5196 ELECTRON MICROSCOPIC STUDIES OF HUMAN PITUITARY TUMORS II. ACIDOPHILIC ADENOMAS. (E.) Schechter, J. (U. Southern California, Sch. Med., Los Angeles). *Am J Anat* 138(3):387-400, 1973.

Electron microscopic studies were made of 9 cases of acidophilic adenoma associated with acromegaly. Numerous somatotrophs were contained in the tumor parenchyma. Many of the somatotrophs appeared normal and were engaged in protein synthesis. Marked variations in the electron-opaque characteristics of the parenchymal cells occurred in half the specimens studied and particularly, although not exclusively, the somatotrophs were affected. Some somatotrophs were characterized by marked electron-opacity of the cytoplasmic matrix and nuclear materials, irregular cell contours, hypertrophied cytomembrane systems frequently containing proteinaceous material, and large amounts of secre-

tory granules. Plasma membranes were disrupted throughout the parenchyma, releasing large numbers of secretory granules and cytoplasmic fragments to pericapillary or extracellular spaces. Follicular cells were rarely observed, but, when present, they contained an abundance of fine cytoplasmic filaments and closely resembled follicular cells described in ACTH-secreting tumors.

- 5197 POLYP CONTROVERSY. (E.) Weiss, O. (U.S. Veterans Admin. Hosp., Lyons, N.J.).
Am J Proctol 24(6):481-485, 1973.

Polyps are defined as adenomas which are actual neoplasms and derive from the colon mucosa. There are two types: simple adenomas, and papillary adenomas or villous polyps. One-third of all polyps show signs of malignancy, with the percentage of malignancy in villous adenomas being higher. The incidence of polyps appears to increase with age, although there are no consistent figures available. Polyps less than 10 mm appear less likely to be malignant, while 24% of polyps over 1.2 cm have been said to be cancerous. Their etiology remains cryptic, although they are more frequent in cigarette smokers and patients with arteriosclerotic heart disease. There is evidence to support the theory of transformation, which states that polyps are premalignant lesions and may eventually turn into carcinomas. This theory has been challenged on the basis that it is based on circumstantial evidence; those who challenge the transformation theory give evidence to support the notion that carcinomas of the colon and rectum develop "de novo". However, recent histochemical and electron microscopic evidence lend more support to the transformation theory. Thus, it appears best to leave no polyp untreated, the choice of treatment remaining a matter of personal judgment.

- 5198 CHOLESTEROL METABOLISM IN RATS BEARING MORRIS HEPATOMA 7777. (E.) Grigor, M. R. (Oak Ridge Associated U., Tenn.), M. L. Blank and F. Snyder. *Cancer Res* 33(8):1870-1874, 1973.

- 5199 FREE AND MEMBRANE-BOUND POLYSOMES IN 3T3 AND Py3T3 CELLS. (E.) Noll, M. (Biol. Ctr., U. Basel, Switzerland) and M. M. Burger. *Experientia* 29(6):777, 1973.

- 5200 QUANTITATIVE DETERMINATION AND LOCATION OF NEWLY SYNTHESIZED VIRUS-SPECIFIC RNA IN CHICKEN CELLS INFECTED WITH ROUS SARCOMA VIRUS. (E.) Parsons, J. T. (U. Zurich, Switzerland), J. M. Coffin, R. K. Haroz, P. A. Bromley and C. Weissmann. *Experientia* 29(6):777, 1973.

- 5201 THE ACTIVATION OF COAGULATION BY EXTRACTS OF MUCUS: A POSSIBLE PATHWAY OF INTRAVASCULAR COAGULATION ACCOMPANYING ADENOCARCINOMAS. (E.) Pineo, G. F. (Royal Postgraduate Med. Sch., London, England), E. Regoeczi, M. W. C. Hatton and M. C. Brain. *J Lab Clin Med* 82(2):255-266, 1973.

- 5202 GIANT LEIOMYOMA OF THE UTERUS: REPORT OF A CASE AND REVIEW OF THE LITERATURE. (E.) Singhabhandhu, B. (Piedmont Hosp., Atlanta, Ga.), J. T. Akin, J. H. Ridley, S. W. Gray and J. E. Skandalakis. *Am Surg* 39(7):391-397, 1973.

- 5203 A REVIEW OF 1,063 BREAST BIOPSIES IN A SERIES OF PRIVATE PATIENTS FROM 1948 THROUGH 1963. (E.) Horwitz, A. (George Washington U. Hosp., Washington, D.C.). *Am Surg* 39(7):372-379, 1973.

- 5204 ONCOCYTOMA OF THE PAROTID GLANDS. CASE PRESENTATION AND REVIEW OF THE LITERATURE. (E.) Hernandez, A. (Wilmington Med. Ctr., Del.) and H. S. Rafal. *Del Med J* 45(8):219-221, 1973.

- 5205 A REVIEW OF PATIENTS WITH CARCINOMA OF THE UTERINE CERVIX - TREATED AT KENT GENERAL HOSPITAL FROM 1961-1971. (E.) Ryan, F. M. (Coll. Med., Pennsylvania St. U., Hershey) *Del Med J* 45(8):221-224, 1973.

- 5206 PRIMARY INTRACRANIAL GERM CELL TUMOURS. (E.) Jellinger, K. (Neurological Inst., U. Vienna, Austria). *Acta Neuropathol (Berl)* 25(4):291-306, 1973.

- 5207 VARIABILITY IN AKR MOUSE LEUKEMIA MORTALITY. (E.) Kohn, R. R. (Inst. Path., Case Western Reserve U., Cleveland, Ohio) and D. Novak. *J Natl Cancer Inst* 51(2):683-685, 1973.

- 5208 CLINICAL EVOLUTION, MORPHOLOGICAL ASPECT AND CYTOGENETICAL STRUCTURE CORRELATIONS IN HUMAN MALIGNANT OVARIAN TUMORS. (E.) Moraru, I. (Cantacuzino Hosp., Bucharest, Rumania), V. Velciu, L. Fadei, C. Budu and E. Badea. *Acta Morphol Acad Sci Hung, Suppl* 14:116, 1973.

- 5209 INTRANUCLEAR MULTITUBULAR STRUCTURES IN ADENOCARCINOMA CELLS OF THE HUMAN ENDOMETRIUM. (E.) Morano, E. (St. Andrea Hosp., Vercelli, Italy), E. Bollero and F. Mazzucchi. *J Submicrosc Cytol* 5(2):153-156, 1973.

- 5210 UPTAKE OF ⁵⁷CO-BLEOMYCIN BY LIVER TUMOR. TUMORSCANNING WITH ⁵⁷CO-BLEOMYCIN. (E.) Maeda, T. (Kyushu Natl. Cancer Ctr. Hosp., Fukuoka, Japan) and M. Tanaka. *Radioisotopes* 22(6):46-48, 1973.

- 5211 SPONTANEOUS TUMOR IN BD II RATS. (Ger.) Kantemir, I. (Pharmacol. Inst. U. Ankara, Turkey). *Arzneim Forsch* 23(6):883-884, 1973.
- 5212 CLINICO-MORPHOLOGICAL CHARACTERISTICS OF THE OATCELL CANCER OF THE LUNG. (Rus.) Blinov, N. N. (N. N. Petrov Res. Inst. Oncology, Leningrad, USSR) and V. G. Rukavishnikova. *Vopr Onkol* 19(6):25-31, 1973.
- 5213 AN ESTIMATION OF BIOCHEMICAL REACTIONS IN DIAGNOSIS OF ABDOMINAL TUMORS. (Rus.) Dzjubko, N. Ya. (Res. Roentgeno-Radiol. Oncol. Inst., Kiev, USSR), A. N. Alferov and N. D. Dumbadze. *Vopr Onkol* 19(6):34-38, 1973.
- 5214 THE KINETICS OF CONTAINING OF SULPHYDRILE AND DISULPHIDE GROUPS AND PROTEIN IN THE PROCESS OF DEVELOPMENT OF EHRICH ASCITIC TUMOR. (Rus.) Romanovsky, I. V. (Inst. Chemical Physics, Moscow, USSR), A. I. Agatova and E. N. Novikova. *Vopr Onkol* 19(6):60-64, 1973.
- 5215 'SYSTEMIC EFFECTS' DURING THE GROWTH OF MALIGNANT EXPERIMENTAL TUMORS. SIGNIFICANCE, OF UNSPECIFIC ORGAN CHANGES IN THE HOST ORGANISM AS 'INHERENT FACTORS' OF THE EXPERIMENT. (E.) Ertl, N. (Heidelberg, Germany). *Oncology* 27(5):415-429, 1973.
- 5216 CORRELATED DECREASE OF MANGANESE AND CATALASE IN LIVER. INVESTIGATIONS IN RATS BEARING YOSHIDA-SARCOMAS AND IN MICE INJECTED WITH TUMOR AND TISSUE EXTRACTS. (Ger.) Zimmerer, J. (German Cancer Res. Ctr., Heidelberg), H. Wesch, K. Wayss, and M. Volm. *Z Krebsforsch* 79(1):39-48, 1973.
- 5217 CARDIAC LIPOMA OF THE INTERATRIAL SEPTUM. (Ger.) Klein, P. J. (Inst. Pathol., U. Cologne, Germany) and H. E. Schaefer. *Z Krebsforsch* 79(1):11-18, 1973.
- 5218 INVESTIGATIONS ON THE BEHAVIOR OF ESSENTIAL TRACE ELEMENTS DURING THE GROWTH OF TUMORS IN RATS. (Ger.) Wesch, H. (German Cancer Res. Ctr., Heidelberg), J. Zimmerer, K. Wayss and M. Volm. *Z Krebsforsch* 79(1):19-30, 1973.
- 5219 SOLID TERATOMAS OF THE OVARY. (E.) Wisniewski, M. (Mount Sinai Sch. Med., City U. New York, N.Y.) and L. M. Deppisch. *Cancer* 32(2):440-446, 1973.
- 5220 SUBCUTANEOUS LEUKAEMIC DEPOSIT IN CHRONIC MYELOGENOUS LEUKAEMIA - COURSE AND HISTOLOGICAL APPEARANCE. (E.) Misra, R. C. (Sch. Tropical Med., Calcutta, India), M. M. Rakshit and A. K. Basu. *J Assoc Physicians India* 21(7):623-625, 1973.
- 5221 THE COEXISTENCE OF AN OVARIAN BRENNER TUMOR AND ENDOMETRIAL STROMAL SARCOMA. (E.) Benisch, B. M. (Mount Sinai Sch. Med., City U. New York, N.Y.) and C. Toker. *Mt Sinai J Med NY* 40(5):689-692, 1973.
- 5222 MELANOMAS OF THE LIMBIC CONJUNCTIVAE. (Fr.) Trottier, M. (Sacred Heart Hosp., Montreal, Canada). *Union Med Can* 102(10):2113-2116, 1973.
- 5223 THE EFFECT OF HYPERTHERMIA ON THE GROWTH OF A HETEROTRANSPLANTED HUMAN SIGMA-CARCINOMA (TUMOR GW-39). (Ger.) Wüst, G. (Med. Clinic, U. Münster/Westphalia, Germany) and L. Prang. *Z Krebsforsch* 79(3):204-211, 1973.
- 5224 *IN VITRO* EFFECT OF HYPERTHERMIA ON THE INCORPORATION RATE OF NUCLEIC ACID PRECURSORS IN TUMORS AND NORMAL TISSUES. (Ger.) Wüst, G. P. (Med. Clinic, U. Münster, Germany), K. Norpoth, U. Witting and W. Oberwittler. *Z Krebsforsch* 79(3):193-203, 1973.
- 5225 BRAIN TUMORS IN CHILDHOOD. (Ger.) Arendt, A. (Path. Inst., Karl-Marx-U., Leipzig, East Germany) and B. Möller. *Arch Geschwulstforsch* 41(2):164-176, 1973.
- 5226 PROPOSAL FOR PATHOLOGICAL CLASSIFICATION OF MAMMARY CARCINOMA ACCORDING TO THE TNM SYSTEM. (Ger.) Berndt, H. (Central Inst. Cancer Res., East Germany Acad. Sci., Berlin), H.-J. Gütz, K.-H. Jacobasch, B. Prah, C.-N. Schremmer, R. Strohwig, G. P. Wildner and G. Wolff. *Arch Geschwulstforsch* 41(2):146-163, 1973.
- 5227 CYTOFLUORIMETRIC STUDY OF THE DNA CONTENT IN GASTRIC CANCER CELLS. (Rus.) Matjushina, E. D. (Res. Inst. Gastroenterology, USSR Ministry Publ. Hlth.), S. I. Rapoport and A. V. Zelenin. *Vopr Onkol* 19(8):8-13, 1973.
- 5228 ULTRASTRUCTURE OF SARCOMA 180. (E.) Zuckerberg, C. (Fac. Med., U. Buenos Aires, Argentina). *Cancer Res* 33(10):2278-2282, 1973.
- 5229 KARYO-CYTOMETRICAL AND CYTOCHEMICAL ANALYSIS IN EROSION AND CANCER OF THE CERVIX UTERI. (Rus.) Tolmachev, V. S. (Kazakh Res. Inst. Oncology, Radiology, Alma-Ata, USSR) and A. I. Shibanova. *Vopr Onkol* 19(7):16-21, 1973.
- 5230 SEX CHROMATIN IN EPITHELIAL CELLS OF CANCEROUS TUMOR OF THE UTERINE CERVIX. (Rus.) Sternjuk, B. P. (Med. Inst., Regional Dispensary, Ljvov, USSR) and D. S. Kastrukova. *Vopr Onkol* 19(7):9-15, 1973.

- 5231 SARCOMA OF THE FALLOPIAN TUBE. (E.)
Blaikley, J. B. (Guy's Hosp., London, England). *J Obstet Gynaecol Br Commonw* 80(8):759-760, 1973.
- 5232 ADVANCED ADENOCARCINOMA OF THE FALLOPIAN TUBE. (E.) Blaikley, J. B. (Guy's Hosp., London, England). *J Obstet Gynaecol Br Commonw* 80(8):757-758, 1973.
- 5233 PRIMARY LEIOMYOSARCOMA OF THE VAGINA. LIGHT AND ELECTRON MICROSCOPIC OBSERVATIONS. (E.) Tobon, H. (U. Pittsburgh, Sch. Med., Pa.), A. I. Murphy and H. Salazar. *Cancer* 32(2):450-457, 1973.
- 5234 COLLAGENOLYTIC ACTIVITY IN MALIGNANT MELANOMA: PHYSICOCHEMICAL STUDIES. (E.)
Yamanishi, Y. (Memphis VA Hosp., Tenn.), E. Maeyens, M. Kh. Dabbous, H. Ohyama and K. Hashimoto. *Cancer Res* 33(10):2507-2512, 1973.
- 5235 HUMAN NEUROBLASTOMA CELL CULTURE: EFFECT OF 5-BROMODEOXYURIDINE ON MORPHOLOGICAL DIFFERENTIATION AND LEVELS OF NEURAL ENZYMES. (E.)
Prasad, K. N. (U. Colorado Med. Ctr., Denver), B. Mandal and S. Kumar. *Proc Soc Exp Biol Med* 144(1):38-42, 1973.
- 5236 A TESTOSTERONE-SECRETING TUMOUR OF THE ADRENAL PRODUCING VIRILISATION IN A FEMALE INFANT. (E.) Burr, I. M. (Vanderbilt U. Sch. Med., Nashville, Tenn.), J. Sullivan, T. Graham, W. H. Hartman and J. O'Neill. *Lancet* (7830):643-644, 1973.
- 5237 A POSSIBLE *IN-VITRO* BLOOD TEST FOR CANCER. (E.) Fish, R. G. (Velindre Hosp., Whitchurch, Cardiff, England). *Lancet* (7830):670, 1973.
- 5238 THE ENZYMIC ACTIVITY OF EXPERIMENTAL UTERINE FIBROMYOMAS. (Rus.) Melnikov, Ju. G. (1st Med. Inst., Moscow, USSR). *Vopr Onkol* 19(7):38-43, 1973.
- 5239 SIGNIFICANCE OF THE SAMPLING TIME IN CHROMOSOME STUDIES IN LEUKAEMIA. (E.) Muldal, S. (Christie Hosp., Manchester, England). *Br J Cancer* 28(2):194-195, 1973.
- 5240 CARCINOGENESIS BY NATURAL SELECTION OF SPECIES. (E.) Cambior, G. J. (Tarragona, Spain). *Lancet* (7833):846-847, 1973.
- 5241 INCIDENCE OF HEAD AND NECK METASTASES FROM GENITO-URINARY NEOPLASMS. (E.) Flocks, R. H. (U. Hosp., Iowa City, Iowa) and D. L. Boatman. *Laryngoscope* 83(9):1527-1539, 1973.
- 5242 EFFECTS OF DIBUTYRYL CYCLIC AMP AND RELATED COMPOUNDS ON NEWT LIMB REGENERATION BLASTEMAS *IN VITRO*. I. ³H-THYMIDINE INCORPORATION. (E.)
Foret, J. E. (Dept. Zoology, U. New Hampshire, Durham) and G. L. Babich. *Oncology* 28(1):83-88, 1973.
- 5243 SUCCINIC ACID DEHYDROGENASE ACTIVITY OF WALKER RAT CARCINOMA 256 WHEN UTILIZING RIBOFLAVIN HOMOLOGS. (E.) Rigenberg, L. K. (Sch. Med., U. Maryland, Baltimore) and J. P. Lambooy. *Proc Soc Exp Biol Med* 143(4):1211-1214, 1973.
- 5244 FAMILIAL OCCURRENCE OF BREAST CANCER. (E.)
Saikkonen, J. (Finspang Hosp., Sweden) and J. Sääf. *Lancet* (7829):626, 1973.
- 5245 TRANSPLANTED MUCOSA OF THE MOUSE INTESTINE AS MODEL FOR CELL PROLIFERATION STUDIES. (E.) Rowiński, J. (Med. Sch., Warsaw, Poland) and M. Kamiński. *Gastroenterology* 65(4):642-646, 1973.
- 5246 MALIGNANT TUMORS OF THE SUBMAXILLARY GLAND. (E.) Byers, P. M. (U. Texas-M. D. Anderson Hosp., Tumor Inst., Houston), R. H. Jesse, O. M. Guillaumondegui and M. A. Luna. *Am J Surg* 126(4):458-463, 1973.
- 5247 FOLLICULAR CARCINOMA OF THE THYROID. (E.)
Tollefsen, H. R. (Mem. Sloan-Kettering Cancer Ctr., New York, N.Y.), J. P. Shah and A. G. Huvos. *Am J Surg* 126(4):523-528, 1973.
- 5248 EFFECTS OF DIBUTYRYL CYCLIC AMP AND RELATED COMPOUNDS ON NEWT LIMB REGENERATION BLASTEMAS *IN VITRO*. ¹⁴C-LEUCINE INCORPORATION. (E.)
Babich, G. L. (Dept. Zoology, U. New Hampshire, Durham) and J. E. Foret. *Oncology* 28(1):89-95, 1973.
- 5249 THE OCCURRENCE OF CARCINOID TUMOUR IN TERATOMA OF THE TESTIS. (E.) Sinnatamby, C. S. (St. Bartholomew's Hosp., London, England), A. B. Gordon and J. D. Griffiths. *Br J Surg* 60(7):576-579, 1973.
- 5250 CONGENITAL MELANOCYTIC NEVI OF THE SMALL AND GARTMENT TYPE. CLINICAL, HISTOLOGIC, AND ULTRASTRUCTURAL STUDIES. (E.) Mark, G. J. (Massachusetts Gen. Hosp., Boston), M. C. Mihm, M. G. Liteplo, R. J. Reed and W. H. Clark. *Hum Pathol* 4(3):395-418, 1973.
- 5251 CALCITONIN- AND ACTH-PRODUCING CELLS IN A CASE OF MEDULLARY CARCINOMA OF THE THYROID. IMMUNOFLOUORESCENCE INVESTIGATIONS. (E.) Bussolati, G. (Inst. Anatomy, Path., Histology, U. Turin, Italy), S. Van Noorden and C. Bordin. *Virchows Arch [Pathol Anat]* 360:123-127, 1973.

- 5252 ORIGIN OF THE RING CHROMOSOME IN A HUMAN RECURRENT MENINGIOMA STUDIED WITH G-BAND TECHNIQUE. (E.) Mark, J. (Huddinge Hosp., Sweden). *Acta Pathol Microbiol Scand* [A] 81(8):588-590, 1973.
- 5253 ALKALINE AND ACID PHOSPHATASE ACTIVITY IN SARCOID LYMPH NODES. (E.) Palva, T. (Dept. Otolaryngology, U. Oulu, Finland), V. Raunio and R. Nousiainen. *Acta Pathol Microbiol Scand* [A] 81(8):577-582, 1973.
- 5254 PITUITARY ADENOMAS AND THE HISTOLOGY OF THE PROSTATE IN ELDERLY MEN. AN ANALYSIS IN AN AUTOPSY SERIES. (E.) Haugen, O. A. (Ullevål Hosp., Oslo, Norway). *Acta Pathol Microbiol Scand* [A] 81(8):425-434, 1973.
- 5255 LOSS OF EPITHELIAL BLOOD GROUP SUBSTANCE A IN ORAL CARCINOMAS. (E.) Dabelsteen, E. (U. Hosp., Copenhagen, Denmark) and J. J. Pindborg. *Acta Pathol Microbiol Scand* [A] 81(8):435-444, 1973.
- 5256 THE RELATIONSHIP OF ERYTHROMONOCYTIC LEUKEMIA TO OTHER MYELOPROLIFERATIVE DISORDERS. (E.) Shaw, M. T. (U. Oklahoma Hlth. Sci. Ctr., Oklahoma City), S. S. Bottomley, R. H. Bottomley and K. K. Hussein. *Am J Med* 55:542-548, 1973.
- 5257 OBSERVATIONS ON A CASE OF LEIOMYOSARCOMA OF THE KIDNEY. (E.) Campo, B. (Dept. Surg., U. Milan, Italy) and P. Rigatti. *Tumori* 59(1):57-62, 1973.
- 5258 PITUITARY ADENOMA WITH SPECIAL REFERENCE TO ITS EXTRASELLAR EXTENSION. (Jap.) Usui, K. (Nagoya U. Sch. Med., Japan), N. Kageyama, O. Sato and M. Furuse. *Brain Nerve* 25(8):1011-1018, 1973.
- 5259 PROGRESSIVE KARYOTYPIC CHANGES *IN VITRO* IN THE EHRlich LETTÉ ASCITES SUBLINE SZEL. (E.) Stephenson, E. M. (Sch. Biol. Sci., U. Sydney, Australia) and N. G. Stephenson. *Eur J Cancer* 9(4):273-279, 1973.
- 5260 DESMOID FIBROBLASTOMA. INTRACITOPLASMIC COLLAGENOSYNTHESIS IN A PECULIAR FIBROBLASTIC TUMOR: LIGHT AND ULTRASTRUCTURAL STUDY OF A CASE. (E.) Allegra, S. R. (Tufts U. Sch. Med., Boston, Mass.) and P. A. Broderick. *Hum Pathol* 4(3):419-429, 1973.
- 5261 DIFFERENCES IN RNAase COMPLEMENTS BETWEEN TISSUES OF A GENETICALLY TUMOROUS STRAIN (THE *NICOTIANA GLAUCA* X *N. LANGSDORFFII* HYBRID) AND ITS NON TUMOROUS PARENTS (*N. GLAUCA* AND *N. LANGSDORFFII*), GROWN *IN VITRO*. (E.) Geri, C. (Natl. Res. Council, Pisa, Italy), R. Parenti and M. Durante. *Ital J Biochem* 22(1):23-35, 1973.
- 5262 OBSERVATIONS ON THE RELATIONSHIP BETWEEN LYMPHOSARCOMA AND HODGKIN'S DISEASE. (E.) Wisniewski, M. (Mount Sinai Sch. Med., City U. New York, N.Y.) and C. Toker. *Mt Sinai J Med NY* 40(5):681-692, 1973.
- 5263 DETERMINATION OF CARCINOMA OF THE CERVIX AND ENDOMETRIUM USING A SINGLE SLIDE. A CASE REPORT. (E.) Gravlee, Jr., L. C. (Gravlee-Wideman Clin., Birmingham, Ala.) and H. J. Lohmann. *J Med Assoc State Ala* 43(3):165, 1973.
- 5264 ZINC IN SERUM AND URINE IN HEMOBLASTOSIS AND MALIGNANCY. (E.) Mikac-Devic, D. (Med. Fac., U. Zagreb, Yugoslavia), N. Milic and H. Stankovic. *Acta Med Jugosl* 26(4):5-10, 1972.
- 5265 ADENOCARCINOMA OF THE LUNG PRESENTING AS A SOLITARY PULMONARY NODULE. (E.) Lawhorne, Jr., T. W. (Johns Hopkins U. Sch. Med., Hosp., Baltimore, Md.), R. R. Baker and D. Carter. *Johns Hopkins Med J* 133(2):82-87, 1973.
- 5266 MEDULLARY CARCINOMA OF THE LUNG WITH AMYLOID STROMA: A COUNTERPART OF MEDULLARY CARCINOMA OF THE THYROID. (E.) Gordon, H. W. (U. California, Sch. Med., Irvine), R. Miller, Jr., C. Mittman. *Hum Pathol* 4(3):431-436, 1973.
- 5267 NEOPLASMS AND PROLIFERATIVE LESIONS IN 1065 NONHUMAN PRIMATE NECROPSIES. (E.) Seibold, H. R. (Tulane U., Delta Reg. Primate Res. Ctr., Covington, La.), and R. H. Wolf. *Lab Anim Sci* 23(4):533-539, 1973.
- 5268 ASSOCIATION OF MALIGNANT MELANOMA AND MALIGNANT LYMPHOMA. (E.) Tashima, C. K. (Saint Francis Hosp., Honolulu, Hawaii). *Lancet* 7823:266, 1973.
- 5269 CALCIUM-PHOSPHATE-PHOSPHOLIPID COMPLEXES IN EXPERIMENTAL TUMORS: THEIR POSSIBLE RELATIONSHIP WITH TUMOR CALCIFICATION. (Ger.) Anghileri, L. J. (Essen Clin., Ruhr-U., West Germany) and R. Dermietzel. *Z Krebsforsch* 79(3):148-156, 1973.
- 5270 MAST CELL DISTRIBUTION IN PERIPHERAL NERVE TUMORS. (Ger.) Justich, E. (Inst. Path. Anatomy, U. Graz, Austria). *Acta Neuropathol (Berl)* 25(4):271-280, 1973.
- 5271 A COMMON CONFORMATIONAL FEATURE IN SEVERAL PROKARYOTIC AND EUKARYOTIC 5 S RNAs: A HIGHLY EXPOSED, SINGLE-STRANDED LOOP AROUND POSITION 40. (E.) Vigne, R. (Ctr. Molec. Biol., Marseille, France), B. R. Jordan and R. Monier. *J Mol Biol* 76(2):303-311, 1973.

- 5272 ZOLLINGER-ELLISON SYNDROME WITH HYPOGLYCEMIA ASSOCIATED WITH CALCIFICATION OF THE TUMOR AND ITS METASTASES. (E.) Bozyski, E. M. (U. North Carolina Sch. Med., Chapel Hill), K. Woodruff and J. T. Sessions, Jr. *Gastroenterology* 65(4):658-661, 1973.
- 5273 LARGE SOLITARY HEPATIC HAMARTOMA. (E.) Orda, R. (Ichilov Hosp., Tel Aviv, Israel), T. Wiznitzer, B. Griffel and J. B. Bawnik. *Am Surg* 39(10):592-595, 1973.
- 5274 PAPILLARY CYSTADENOMA OF KIDNEY. (E.) Loening, S. (VA Hosp., White River Junction, Vermont) and J. R. Richardson. *J Urol* 1(6):593-595, 1973.
- 5275 THE BIOCHEMICAL ENVIRONMENT OF THE MAMMALIAN NUCLEUS. (E.) Siebert, G. (U. Hohenheim, Stuttgart, Germany). *Sub-Cell Biochem* 1:277-292, 1972.
- 5276 METASTATIC THYROID CARCINOMA. (E.) Shelley, W. B. (U. Pennsylvania Sch. Med., Philadelphia), H. Beerman and H. T. Enterline. *J Am Med Assoc* 226(2):173-174, 1973.
- 5277 REPORT OF A CHORDOMA PRESENTING JUGULAR FORAMEN SYNDROME AS THE INITIAL SIGNS AND SYMPTOME. (Jap.) Nonaka, F. (Brain Inst., Niigata U., Japan), S. Sato, G. Uemura and Y. Furusawa. *Brain Nerve* 25(8):1075-1079, 1973.
- 5278 FURTHER DATA ON THE COMMON ORIGIN OF VARIOUS STEM-LINES IN HUMAN TUMORS. (E.) Olinici, C. D. (Inst. Oncology, Cluj, Romania). *Cytologia (Tokyo)* 38(2):271-276, 1973.
- 5279 ROLE OF DNA TOPOLOGY IN TRANSCRIPTION OF COLIPHAGE λ *IN VIVO* II. DNA TOPOLOGY PROTECTS THE TEMPLATE FROM EXONUCLEASE ATTACK. (E.) Pilarski, L. M. (U. Adelaide, Australia) and J. B. Egan. *J Mol Biol* 76(2):257-266, 1973.
- 5280 DIAGNOSIS OF MENINGEAL LEUKEMIA IN ACUTE CHILDHOOD LYMPHOCYTIC LEUKEMIA WITH PERIODIC ACID-SCHIFF REACTION OF CYTOCENTRIFUGED LIQUOR. (E.) Feldges, A. J. (St. Jude Children's Res. Hosp., Memphis, Tenn.). *Acta Haematol* 49(3):154-158, 1973.
- 5281 A CLINICAL EXAMINATION ON VALUE DETERMINATION OF THE TNM-CLASSIFICATION OF THE CARCINOMA OF THE BUCCAL CAVITY. (E.) Spiessl, B. (Dept. Surg., U. Basel, Switzerland), J. von Albert, K. Bitter, W. Busch, H. von Domarus, D. Gasser, H. Grasser, W. Hahn, J. E. Hausamen, H. Koch, H. Mehnert, H. Meissel, H. D. Pape, J. Prein, F. Schröder, P. Schulz, W. Steinhilber and E. Waldhart. *Z Krebsforsch* 80(1):83-96, 1973.
- 5282 PRIMARY TRANSITIONAL CELL CARCINOMA OF THE PROSTATE. (E.) Greene, L. F. (Mayo Clinic Rochester, Minn.), J. J. Mulcahy, M. M. Warren and M. B. Dockerty. *J Urol* 110(2):235-237, 1973.
- 5283 BONE MARROW CALCIUM IN CANCER OF PROSTATE AND BLADDER. (E.) Megalli, M. R. (Columbia U. Coll. Physicians, Surgeons, New York, N.Y.), E. Gursel, L. Rudin and R. J. Veenema. *J Urol* 2(1):25-27, 1973.
- 5284 HISTOCHEMICAL CHARACTERISTICS OF PARAFOLLICULAR CELLS AND MEDULLARY THYROID CARCINOMA. (E.) DeLellis, R. A. (Boston U. Sch. Med., Mass.) and K. Balogh. *Am J Pathol* 72(1):119-126, 1973.
- 5285 RENAL-CELL CARCINOMA IN CHILDREN. (E.) Ward, J. S. (U. Utah Coll. Med., Salt Lake City) and R. G. Middleton. *J Urol* 2(1):50-52, 1973.
- 5286 INTERSTITIAL-CELL TUMOR OF TESTICLE IN CHILDREN. (E.) Naranjo, C. A. (West Virginia U. Sch. Med., Morgantown), S. J. Kandzari and W. G. Klingberg. *J Urol* 2(1):58-60, 1973.
- 5287 DERMATOGLYPHICS IN LEUKEMIA. (E.) Wertelecki, W. (Med. U. South Carolina, Charleston), C. C. Plato, J. F. Fraumeni and J. D. Niswander. *Pediatr Res* 7(7):620-626, 1973.
- 5288 MALIGNANT HAEMANGIO-ENDOTHELIOMA ASSOCIATED WITH CONGENITAL ADRENAL HYPERPLASIA. (E.) Donald, D. (Dept. Path., U. Aberdeen, Scotland). *J Pathol* 109(4):361-363, 1973.
- 5289 WHITE BLOOD CELL ACID HYDROLASES IN LEUKAEMIAS, MUCOPOLYSACCHARIDOSES AND MANNOSIDOSIS. (E.) Hultberg, B. (U. Hosp., Lund, Sweden), S. Autio, B. Berg and P. A. Öckerman. *Scand J Haematol* 10(4):265-272, 1973.
- 5290 RELATIONSHIP OF THE PRE-TREATMENT PERIPHERAL LYMPHOCYTE COUNT TO HISTOLOGY IN HODGKIN'S DISEASE. (E.) Henry, L. (Sheffield Natl. Ctr. Radiotherapy, England), J. Knowelden and H. T. Swan. *Br J Haematol* 24(6):773-776, 1973.
- 5291 FAMILIAL PHEOCHROMOCYTOMA. CASE REPORT AND REVIEW OF THE LITERATURE. (E.) Funyu, T. (Hirosaki U. Sch. Med., Japan), Y. Shiraiwa, K. Nigawara, S. Kudo and T. Mikuni. *J Urol* 110(2):151-154, 1973.
- 5292 RESYNCHRONIZATION OF RNA SYNTHESIS BY COLIPHAGE Φ B REPLICASE AT AN INTERNAL SITE OF THE RNA TEMPLATE. (E.) Kolakofsky, D. (Inst. Molec. Biol., Zurich, Switzerland), M. A. Billeter, H. Weber, and C. Weissmann. *J Mol Biol* 76(2):271-284, 1973.

- 5293 HUMORAL SIMILARITIES OF CARCINOID TUMORS AND MEDULLARY CARCINOMAS OF THE THYROID. (E.) Kaplan, E. L. (U. Chicago, Pritzker Sch. Med., Ill.), G. W. Sizemore, G. W. Peskin and B. M. Jaffe. *Surgery* 74(1):21-29, 1973.
- 5294 FIBRINOLYTIC ACTIVITY IN NORMAL AND CANCEROUS TISSUES OF THE BLADDER. (E.) Hisazumi, H. (Sch. Med., Kanazawa U., Japan), K. Naito, and T. Misaki. *Invest Urol* 11(1):28-34, 1973.
- 5295 ULTRASTRUCTURE OF A RENIN-SECRETING JUXTAGLOMERULAR CELL TUMOR OF THE KIDNEY. (E.) MacCallum, D. K. (Dental Res. Inst., U. Michigan, Ann Arbor.), J. W. Conn, and B. L. Baker. *Invest Urol* 11(1):65-74, 1973.
- 5296 BLADDER CANCER. (E.) Miller, L. S. (U. Texas M. D. Anderson Hosp. Tumor Inst. Houston) *Cancer Bull* 25(3):57-60, 1973.
- 5297 CARCINOMA OF THE PENIS. (E.) Johnson, D. E. (U. Texas M. D. Anderson Hosp. Tumor Inst., Houston). *Cancer Bull* 25(3):50-52, 1973.
- 5298 MALIGNANT MIXED TUMOR OF THE GALLBLADDER. (E.) Higgs, W. R. (Baylor Coll. Med., Houston, Tex.), E. E. Mocega and P. H. Jordan. *Cancer* 32(2):471-475, 1973.
- 5299 BENIGN LIPOBLASTOMATOSIS. ANALYSIS OF 35 CASES. (E.) Chung, E. B. (Howard U., Coll. Med., Washington, D.C.) and F. M. Enzinger. *Cancer* 32(2):482-492, 1973.
- 5300 RETINOBLASTOMA: CHROMOSOME BANDING IN PATIENTS WITH HERITABLE TUMOUR. (E.) Ladda, R. (Mass. Gen. Hosp., Boston), L. Atkins, J. Littlefield and R. Pruett. *Lancet* 2(7827):506, 1973.
- 5301 UNUSUAL MANIFESTATIONS OF RENAL CARCINOMA. A REVIEW OF THE LITERATURE. (E.) Tveter, K. J. (U. Hosp., Akershus, Norway). *Acta Chir Scand* 139(4):401-409, 1973.
- 5302 BENIGN AND MALIGNANT MUCOCELE OF THE APPENDIX. HISTOLOGICAL TYPES AND PROGNOSIS. (E.) Aho, A. J. (Dept. Surg., Path. Anat., U. Turku, Finland), R. Heinonen and P. Lauren. *Acta Chir Scand* 139(4):392-400, 1973.
- 5303 A CLINICAL EVALUATION OF GALLIUM-67 CITRATE SCANNING. (E.) Littenberg, R. L. (Div. Nuclear Med., U. California, San Diego), N. P. Alazraki, R. M. Taketa, R. Reit, S. E. Halpern and W. L. Ashburn. *Surg Gynecol Obstet* 137(3):424-430, 1973.
- 5304 TNM-CLASSIFICATION OF MALIGNANT TUMORS. (Ger.) Harmer, M. H. (Internatl. Union Against Cancer, Geneva, Switzerland). *Arch Geschwulstforsch* 41(4):373-381, 1973.
- 5305 INFLUENCE OF PREIRRADIATION OF LUNG ON DEVELOPMENT OF ARTIFICIAL PULMONARY METASTASES OF FIBROSARCOMA IN MICE. (E.) Withers, H. R. (U. Texas M.D. Anderson Hosp., Tumor Inst., Houston) and L. Milas. *Cancer Res* 33(8):1931-1936, 1973.
- 5306 DEPENDENCE OF 5-METHYLTETRAHYDROFOLATE UTILIZATION BY L5178Y MURINE LEUKEMIA CELLS *IN VITRO* ON THE PRESENCE OF HYDROXYCOBALAMIN AND TRANSCOBALAMIN II. (E.) Chello, P. L. (Yale U. Sch. Med., New Haven, Conn.) and J. R. Bertino. *Cancer Res* 33(8):1898-1904, 1973.
- 5307 DEMONSTRATION OF A GLUCOCORTICOID HORMONE-RECEPTOR COMPLEX IN THE CYTOPLASM OF A HORMONE-RESPONSIVE TUMOUR. (E.) Gardner, D. G. (U. Rochester Sch. Med., Dentistry, N.Y.) and J. L. Wittliff. *Br J Cancer* 27(6):441-444, 1973.
- 5308 CHANGING PATTERNS OF INFECTION IN CANCER PATIENTS. (E.) Gaya, H. (Royal Post-graduate Med. Sch., London, England), M. H. N. Tattersall, R. M. Hutchinson and A. S. D. Spiers. *Eur J Cancer* 9(6):401-406, 1973.
- 5309 ENZYME ACTIVITIES IN INDUCED AND SERIALY TRANSPLANTED MURINE MAMMARY ADENOCARCINOMAS. (E.) Abraham, S. (Children's Hosp. Med. Ctr., Oakland, Calif.), J. Bartley, K. B. DeOme, L. J. Faulkin, Jr. and D. Medina. *J Natl Cancer Inst* 51(1):251-256, 1973.
- 5310 INHIBITION OF HUMAN RHABDOMYOSARCOMA-CELL GROWTH IN AGAR BY DIBUTYRYL CYCLIC AMP. (E.) Sandor, R. (Children's Hosp. Los Angeles, Calif.). *J Natl Cancer Inst* 51(1):257-260, 1973.
- 5311 ELECTRON MICROSCOPIC OBSERVATIONS ON A PAROTID ONCOCYTOMA. (E.) Kay, S. (Med. Coll. Virginia, Richmond) and W. J. S. Still. *Arch Pathol* 96(3):186-188, 1973.
- 5312 SUPPRESSION OF LYMPHOMA DEVELOPMENT IN TETRAPARENTAL AKR MOUSE CHIMAERAS DERIVED FROM OVUM FUSION. (E.) Barnes, R. D. (Clin. Res. Ctr., Harrow, Middlesex, England), M. Tuffrey and C. E. Ford. *Nature [New Biol]* 244(139):282-284, 1973.
- 5313 ISOENZYMIC SPECTRUM OF SERINE TRANSOXY-METHYLASE OF THE LIVER AND HEPATOMAS. (Rus.) Gorjukhina, T. A. (N. N. Petrov Res. Inst. Oncology, USSR Ministry Publ. Hlth., Leningrad) and S. B. Lebedeva. *Vopr Onkol* 19(5):62-66, 1973.

- 5314 ANALYTICAL STRUCTURE EXAMINATION OF CANCER LITERATURE ON THE BASIS OF SABIR-C. (Ger.) Sandor, L. (German Cancer Res. Ctr., Heidelberg), G. Wagner and M. Wolff-Terroine. *Z Krebsforsch* 80(1):69-82, 1973.
- 5315 EVALUATION OF PHA-P REACTIVITY OF LYMPHOCYTES IN TUMOR DEVELOPMENT BY ACID PHOSPHATASE ACTIVITY. (E.) Gillissen, G. (Fac. Med., Technical U., Aachen, Germany), P. Mecke and E. Mecke. *Z. Krebsforsch* 80(1):45-52, 1973.
- 5316 THE INFLUENCE OF SEX AND AGE OF TUMOURS ON THE KARYOTYPE OF AN EHRlich ASCITES-CARCINOMA OF MOUSE. (Ger.) Keutsch, F. (Radio-biol. Inst. U. Zurich, Switzerland) and H. Fritz-Niggli. *Z Krebsforsch* 80(1):53-68, 1973.
- 5317 ISOLATION AND CHARACTERIZATION OF POKEWEED MITOGEN-LIKE PHYTOMITOGENS FROM SHORIKU, *PHYTOLACCA ESCULENTA*. (E.) Tokuyama, H. (Cancer Res. Inst., Kanazawa U., Takaramachi, Japan). *Biochim Biophys Acta* 317(2):338-350, 1973.
- 5318 GESTATIONAL CHORIOCARCINOMA OF THE TUBE AND OVARY. (E.) Patton, G. W. (Harvard Med. Sch., Boston, Mass.) and D. P. Goldstein. *Surg Gynecol Obstet* 137(4):608-612, 1973.
- 5319 INFECTION AND CANCER: OLD FRIENDS. (E.) DeVita, V. J. (Nat'l. Cancer Inst., Bethesda, Md.) and R. C. Young. *Annals Intern Med* 79(4):597-599, 1973.
- 5320 NUCLEAR MAGNETIC RESONANCE STUDIES OF SEVERAL EXPERIMENTAL AND HUMAN MALIGNANT TUMORS. (E.) Hollis, D. P. (Johns Hopkins U. Sch. Med., Baltimore, Md.), J. S. Economou, L. C. Parks, J. C. Eggleston, L. A. Saryan and J. L. Czeisler. *Cancer Res* 33(9):2156-2160, 1973.
- 5321 INCIDENCE OF CARCINOMA IN COLD NODULES OF THE THYROID GLAND. (E.) Messaris, G. (Dept. Surg., U. Athens, Greece), G. N. Evangelou and C. Tountas. *Surgery* 74(3):447-448, 1973.
- 5322 SYNTHESIS AND RELEASE OF PARATHYROID HORMONE BY A RENAL CARCINOMA IN CELL CULTURE. (E.) Greenberg, P. B. (Dept. Med., U. Melbourne, Australia), T. J. Martin and H. S. Sutcliffe. *Clin Sci Molec Med* 45(2):183-191, 1973.
- 5323 HISTOPATHOLOGY OF PLASMA CELL TUMORS. (Fr.) Mazabraud, A. (No affiliation). *Bull Cancer (Paris)* 59(4):363-365, 1972.
- 5324 CONGENITAL LEUKEMIA ASSOCIATED WITH MONOGOLISM. (It.) De Ritis, L. (Munic. Hosp., Latisana, Italy) and G. Della Porta. *Minerva Pediatr* 25(12):563-567, 1973.
- 5325 ULTRASTRUCTURE OF TUMORS OF THE ADRENAL CORTEX IN CUSHING'S SYNDROME. (Ger.) Mitschke, H. (Pathol. Inst., U. Hamburg, Germany), W. Saeger and H. J. Breustedt. *Virchows Arch (Pathol Anat)* 360(3):253-264, 1973.
- 5326 ROLE OF THE HYPOTHALAMUS IN THE DEVELOPMENT OF EXPERIMENTAL TUMORS IN ALBINO RATS. (Rus.) Artiunian, R. K. (No affiliation), R. A. Gabrielian and S. R. Tokhian. *Biol Zh Armenii* 26(3):91-93, 1973.
- 5327 STUDY OF SEXUAL CHROMATIN AS ANOTHER SIGN OF THE BIOLOGICAL ACTIVITY OF BREAST CANCER. (Rus.) Ganina, K. P. (Inst. Oncol. Problems, Acad. Sci. Ukr. SSR, USSR), L. P. Lysiuk and L. Z. Popishchuk. *Lab Delo* (5):266-268, 1973.
- 5328 BENIGN LIVER CELL TUMORS. CLASSIFICATION AND ULTRASTRUCTURAL PATHOLOGY. (E.) Phillips, M. J. (Toronto Gen. Hosp., Canada), B. Langer, R. Stone, M. M. Fisher and S. Ritchie. *Cancer* 32(2):463-470, 1973.
- 5329 *IN VITRO* CHROMOSOMAL RADIOSENSITIVITY IN "CHROMOSOMAL BREAKAGE SYNDROMES". (E.) Higurashi, M. (Fac. Med., U. Tokyo, Japan) and P. E. Conen. *Cancer* 32(2):380-383, 1973.
- 5330 THE ISOLATION AND CHARACTERIZATION OF GALLIUM-BINDING GRANULES FROM SOFT TISSUE TUMORS. (E.) Brown, D. H. (Med. Div., Oak Ridge Associated U., Tenn.), D. C. Swartzendruber, J. E. Carlton, B. L. Byrd and R. L. Hayes. *Cancer Res* 33(9):2063-2067, 1973.
- 5331 VARIATIONS AMONG SUBLINES OF INBRED AKR MICE. (E.) Acton, R. T. (California Inst. Technology, Pasadena), E. P. Blankenhorn, T. C. Douglas, R. D. Owen, J. Hilgers, H. A. Hoffman and E. A. Boyse. *Nature [New Biol]* 245(140):8-10, 1973.
- 5332 MEDIASTINAL HYPERFUNCTIONING PARATHYROID TUMORS: REVIEW OF 14 CASES. (E.) Scholz, D. A. (Mayo Clin., Rochester, Minn.), D. C. Purnell, L. B. Woolner and O. T. Clagett. *Ann Surg* 178(2):173-178, 1973.
- 5333 PRIMARY SEBACEOUS CARCINOMA OF THE PAROTID GLAND. (E.) Akhtar, M. (Albert Einstein Med. Ctr., Philadelphia, Pa.), T. G. Gosalbez and H. Brody. *Arch Pathol* 96(3):161-163, 1973.
- 5334 PROLACTIN, PHENOTHIAZINES, ADMISSION TO MENTAL HOSPITAL, AND CARCINOMA OF THE BREAST. (E.) Brugmans, J. (Janssen Pharmaceutica, Beerse, Belgium), F. Verbruggen, J. Dom and V. Schuermans. *Lancet* 2(7827):502-503, 1973.

- 5335 FORAMEN MAGNUM TUMORS. PITFALLS IN DIAGNOSIS. (E.) Howe, J. R. (U. Michigan Med. Ctr., Ann Arbor) and J. A. Taren. *JAMA* 225(9):1061-1066, 1973.
- 5336 SERUM DOPAMINE- β -HYDROXYLASE ACTIVITY IN PATIENTS WITH LEUKEMIA AND IN PATIENTS WITH HEPATOMA. (E.) Goldstein, M. (New York U. Med. Ctr., N.Y.), L. S. Freedman, M. Roffman and L. Helson. *Eur J Cancer* 9(3):233-235, 1973.
- 5337 RECURRENT PARATHYROID ADENOMA. ASSOCIATION WITH PROLONGED THIAZIDE ADMINISTRATION. (E.) Balizet, L. (Highlands Clin., Williamson, W. Va.). *JAMA* 225(10):1238-1239, 1973.
- 5338 POLYMORPHISM OF HUMAN LEUKEMIC CELLS RIBONUCLEASE. (Rus.) Shlyakhovenko, V. A. (Inst. Oncological Problems, Ukrainian SSR Acad. Sci., Kiev, USSR), G. Z. Negrey and M. A. Berman. *Vopr Onkol* 19(5):53-58, 1973.
- 5339 RENAL HAMARTOMAS AND NEPHROBLASTOMATOSIS WITH FETAL GIGANTISM: A FAMILIAL SYNDROME. (E.) Perlman, M. (Soroka Med. Ctr., Beer Sheva, Israel), G. M. Goldberg, J. Bar-Ziv and G. Danovitch. *J Pediatr* 83(3):414-418, 1973.
- 5340 A CALCIFIED ALDOSTERONE-PRODUCING TUMOR IN A HYPERTENSIVE, NORMOKALEMIC, PREPUBERTAL GIRL. (E.) Kelch, R. P. (Dept. Pediatrics, Med., U. California, San Francisco), M. H. Connors, S. L. Kaplan, E. G. Biglieri and M. M. Grumbach. *J Pediatr* 83(3):432-437, 1973.
- 5341 PRIMARY CARCINOMA OF THE FRONTAL SINUS. (Rus.) Rusanova, N. I. (Leningrad Pediatric Med. Inst., USSR). *Vopr Onkol* 19(5):95-96, 1973.
- 5342 PRIMARY CARCINOMA OF THE GASTRIC STUMP. (Sp.) Berenguer, J. (La Fe Munic. Sanit., Valencia, Spain), J. M. Soto, A. Belda, P. Gonzalez-Vara and J. M. Rayon. *Rev Esp Enferm Apar Dig* 40(4):369-378, 1973.
- 5343 UROGRAPHIC DIAGNOSIS OF LEUKEMIA IN A CHILD. CONSIDERATION OF THE ANATOMICAL AND RADIOGRAPHIC PICTURE OF THE LEUKEMIA KIDNEY. (It.) Riggio, S. (Aiuto Materno Provinciale Pediatric Hosp., Rimini, Italy) and M. Papa. *Minerva Pediatr* 25(17):760-764, 1973.
- 5344 CORRELATIONS BETWEEN THE HISTOPATHOLOGY AND NATURAL HISTORY OF INTESTINAL TYPE ADENOCARCINOMA AND DIFFUSE UNDIFFERENTIATED GASTRIC CARCINOMA. (It.) Pilotti, S. (Natl. Inst. Study Treatment Tumors, Milan, Italy), F. Rilke and M. Del Vecchio. *Tumori* 59(3):193-218, 1973.
- 5345 VERY LATE MALIGNANT TRANSFORMATION OF A PAPULAR RETICULOSIS WITH A PROLONGED COURSE. (Fr.) Dupont, A. (St. Pierre Hosp., Louvain, Belgium). *Ann Dermatol Syphiligr (Paris)* 100(2):141-146, 1973.
- 5346 PLASMACYTOMAS OF THE UPPER RESPIRATORY AND GASTROINTESTINAL TRACTS. (Fr.) Ennuyer, A. (Curie Fdn., Paris, France), P. Bataini and G. Chavanne. *Bull Cancer (Paris)* 59(4):389-394, 1972.
- 5347 HISTOLOGICAL ENZYMOLOGY OF SKIN TUMORS IN MAN: RELATIONS BETWEEN THE EPITHELIUM AND STROMA. (Fr.) Dutu, R. (Inst. Oncol., Bucharest, Rumania), M. Nedelea, G. Veluda and C. Longhin. *Ann Histochim* 18(2):159-170, 1973.
- 5348 BONE METASTASES FROM CUTANEOUS EPITHELIOMAS: ONE CASE. (Fr.) Michel, P.-J. (No affiliation), R. Parthiot, C. Masson and B. Cassin. *Ann Dermatol Syphiligr (Paris)* 100(2):147-157, 1973.
- 5349 METASTATIC SPREAD OF EPIDERMOID EPITHELIOMAS OF THE UPPER RESPIRATORY AND GASTROINTESTINAL TRACTS: RESULTS FROM THE AUTOPSY OF 220 PATIENTS. (Fr.) Brugere, J. (Gustave Roussy Inst., Villejuif, France), R. Blache and Y. Cachin. *Bull Cancer (Paris)* 59(4):435-448, 1972.
- 5350 CHROMOSOME STUDY OF 20 EMBRYONIC TUMORS AFTER SHORT-TERM CULTURE. (Fr.) Rousseau, M. F. (Necker Hosp., Paris, France). *Biomedicine* 19(6):275-280, 1973.
- 5351 MESONEPHROID TUMORS (OR CLEAR CELL TUMORS) OF THE OVARY: ANATOMICAL AND CLINICAL STUDY OF TWELVE CASES. (Fr.) Genton, C. (Inst. Pathol. Anat., Zurich, Switzerland). *Schweiz Med Wochenschr* 103(31):1093-1098, 1972.
- 5352 ELECTROPHORETIC STUDY OF PROTEINS IN THE CEREBROSPINAL FLUID IN 104 PATIENTS WITH NEURAXIAL TUMORS. (Fr.) Castaigne, P. (Salpêtrière Hosp., Paris, France), F. Lhermitte, E. Schuller, J.-J. Perrin and N. Delasnerie. *Rev Neurol (Paris)* 127(5):505-515, 1972.
- 5353 CONCENTRATIONS OF CALCITONIN AND CATECHOLAMINES IN PHEOCHROMOCYTOMAS, A MUCOSAL NEUROMA AND MEDULLARY THYROID CARCINOMA. (E.) Voelkel, E. F. (Harvard Sch. Dental Med., Boston, Mass.), A. H. Tashjian, Jr., F. F. Davidoff, R. B. Cohen, C. P. Perlia and R. J. Wurtman. *J Clin Endocrinol Metab* 37(2):297-307, 1973.
- 5354 PRIMARY SARCOMA OF THE GALLBLADDER. (E.) Carpentier, Y. (St.-Pierre U. Hosp., Brussels, Belgium) and J. P. Lambilliotte. *Cancer* 32(2):493-497, 1973.

- 5355 RESULTS FROM ORGAN CULTURE OF HUMAN GASTRIC AND RECTAL CANCERS. (Rus.) Tsytkin, L. B. (Inst. Poliomyelitis Viral Encephalitis, Moscow, USSR) M. K. Voroshilova, A. G. Goriunova and I. K. Lavrova. *Arkhh Patol* 35(1):25-31, 1973.
- 5356 LYMPHOCYTIC BETA-GLUCURONIDASE IN THE LYMPHOPROLIFERATIVE SYNDROMES. (E.) Zittoun, R. (Hotel Dieu, Paris, France), M. Cadiou, C. Dao, J. M. Blanc and J. Bousser. *Biomedicine* 18(5):415-420, 1973.
- 5357 THERMOGENIC ACTIVITY OF BREAST CANCERS. II CHANGES DURING HORMONE TESTS. (Fr.) Gautherie, M. (Fac. Med., Strasbourg, France), C. Gros, P. Bourjat and Y. Quenneville. *Biomedicine* 18(5):421-428, 1973.
- 5358 CHROMOSOME ANALYSIS OF MALIGNANT TUMORS: TECHNIQUE FOR EXAMINING TUMORS IN AN ORGANOTYPIC CULTURE. (Fr.) Carpentier, S. (Fac. Med., Paris, France) and J. Lejeune. *Pathol Biol (Paris)* 21(6):665-669, 1973.
- 5359 PRIMARY LIPOSARCOMAS OF THE BONE: 5 CASES AND REVIEW OF THE LITERATURE. (Fr.) Mandard, J. C. (Fac. Med. Pharm., Caen, France), A. M. Mandard and Y. Le Gal. *Ann Anat Pathol (Paris)* 18(3):329-346, 1973.
- 5360 RENAL CARCINOMA WITH INCREASED ERYTHROPOIETIN PRODUCTION AND SECONDARY POLYCYTHEMIA. (E.) Kvarstein, B. (Riks Hosp., Oslo, Norway), R. Lindemann and W. Mathisen. *Scand J Urol Nephrol* 7(2-3):178-180, 1973.
- 5361 ULTRASTRUCTURE OF ASHKENAZI CELL THYROID ADENOMA. (Rus.) Dmitrieva, N. P. (Inst. Developmental Biol., Moscow, USSR) and V. F. Solomina. *Vopr Onkol* 19(7):3-9, 1973.
- 5362 PRESENCE OF A MITOGENIC FACTOR IN SOLUBLE EXTRACTS FROM TUMORS OF THE COLON. (Fr.) Remacle-Bonnet, M. (UER Med. Marseille, France), S. Kaplanski and R. Depieds. *C R Acad Sci (Paris)* 277(6):599-601, 1973.
- 5363 CYTOCHEMICAL FEATURES OF MALIGNANT MESOTHELIOMAS. (ONE CASE REPORT). (Rus.) Kartavenko, N. B. (Ukrainian Inst. Postgrad Med., USSR) *Vopr Onkol* 19(5):93-94, 1973.
- 5364 ELECTRON MICROSCOPIC STUDIES OF "BASOPHILIC" SECRETION GRANULES OCCURRING IN HYPOPHYSEAL TUMOR CELLS IN CUSHING'S DISEASE. (Fr.) Olivier, L. (Natl. Res. Council Histol. Embryol. Lab., Paris, France), E. Vila-Porcile, F. Peillon and J. Racadot. *C R Soc Biol (Paris)* 166(12):1591-1595, 1972.
- 5365 THE SEROTONIN CONTENT IN BLOOD OF PATIENTS WITH PULMONARY CANCER. (Rus.) Bulygina, A. V. (Altai Med. Inst., U.S.S.R.). *Vopr Onkol* 19(8):42-44, 1973.
- 5366 MESOTHELIOMA AS PRIMARY TUMOR OF THE PERITONEUM. A CASE REPORT. (E.) D'Ambrosio, V. (Overlook Hosp., Summit, N.J.) and M. Hill. *J Med Soc NJ* 70(9):637-639, 1973.
- 5367 ANGIOFOLLICULAR HYPERPLASIA OF LYMPH NODES. (Rus.) Yakovleva, I. A. (Res. Inst. Oncology, Moldavian SSR Ministry Publ. Hlth., Kishinev, USSR), I. F. Korchmaru and A. M. Paraskova. *Vopr Onkol* 19(5):18-24, 1973.
- 5368 THE GENETIC ORIGIN OF LEUCOCYTIC MUCOPOLYSACCHARIDES IN CANCER PATIENTS. (E.) Riesco, A. (Natl. Hlth. Service, Chile) and R. C. Coke. *Br J Cancer* 28(3):269-274, 1973.
- 5369 PRIMARY NEOPLASMS OF THE FALLOPIAN TUBES. (Fr.) Moutquin, J. M. (Hotel Dieu, Montreal, Canada), J. P. Dery and Y. Boivin. *Union Med Can* 102(8):1664-1669, 1973.
- 5370 UNUSUAL PRESENTATION OF HODGKIN'S DISEASE: MASSIVE INVASION OF THE BLOOD BY ABNORMAL CELLS. (Fr.) Thout, C. (St. Luc Hosp., Montreal, Canada), G. Gariepy, M. Monte and L. Perron. *Union Med Can* 102(8):1675-1678, 1973.
- 5371 KAPOSI'S SARCOMA ASSOCIATED WITH PRIMARY AMYLOIDOSIS AND CHRONIC RENAL INSUFFICIENCY. (Fr.) Favreau, J. (Maisonneuve-Rosemont Hosp., Montreal, Canada), L. P. Legresley and C. Beaudry. *Union Med Can* 102(8):1683-1686, 1973.
- 5372 BILATERAL CARCINOMA OF THE BREAST. (E.) Wilson, N. D. (St. Vincent Hosp., Portland, Oregon) and R. E. Alberty. *Am J Surg* 126(2):244-248, 1973.
- 5373 ULTRASTRUCTURAL FEATURES OF MENINGIOMA CELLS. (Fr.) Humeau, C. (Montpellier Sch. Med., France), P. Sentein and B. Vlahovitch. *C R Soc Biol (Paris)* 166(12):1728-1730, 1972.
- 5374 MALIGNANT NEOPLASMS IN CHILDREN. (Rus.) Gazarian, E. S. (Inst. Roentgenol. Oncol., Erevan, USSR), K. S. Saiadian, and V. I. Alekseeva. *Vopr Onkol* 19(6):112-113, 1973.
- 5375 PRIMARY MALIGNANT TUMOR OF THE HEART. (Rus.) Agapova, E. N. (Kuban Med. Inst., Krasnodar, USSR), O. V. Krutovskaia and A. M. Tkachenko. *Vrach Delo* (6):45-47, 1973.

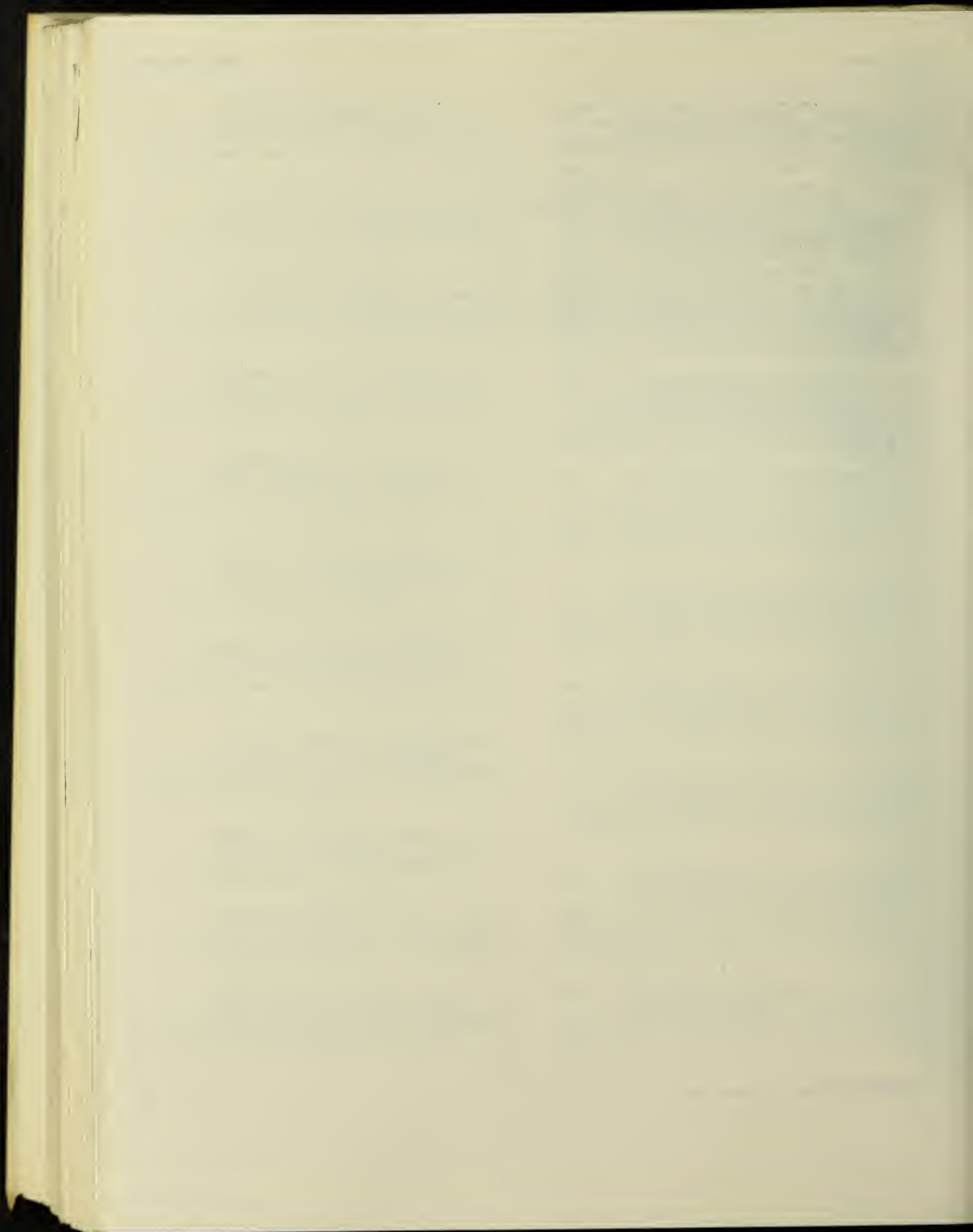
- 5376 NUTRITIONAL AND HORMONAL FACTORS IN THE ETIOLOGY OF ENDOMETRIAL CANCER. (Fr.) Petit, J.-C. (Paus Strauss Ctr., Strasbourg, France), T. H. Klein and J. Herdly. *Bull Cancer (Paris)* 60(1): 71-82, 1973.
- 5377 HISTOPATHOLOGICAL OBSERVATIONS ON NEVI IN CHILDHOOD. (It.) Guarina, M. (G. Gaslini Inst., Genoa, Italy), G. Romagnoli and A. Rizzo. *Minerva Pediatr* 25(14):649-671, 1973.
- 5378 EXTRAMEDULLARY PLASMACYTOMA OF THE RESPIRATORY TRACT. (Cz.) Pellant, A. (Med. Fac., Charles U., Hradec Kralove, Czechoslovakia) and I. Hybasek. *Cesk Otolaryngol* 22(2):108-110, 1973.
- 5379 OCCURRENCE OF GIANT CELL TUMORS OF THE BONE IN OTOLARYNGOLOGY. (Cz.) Hlavacek, V. (Med. Fac., Charles U., Prague, Czechoslovakia). *Cesk Otolaryngol* 22(2):77-83, 1973.
- 5380 ULTRASTRUCTURE AND *IN VITRO* BEHAVIOR OF CEREBRAL SARCOMAS. (It.) Gullotta, F. (Inst. Neuropathol., Univ. Bonn, Germany) and Gl. Kersting. *Acta Neurol (Napoli)* 28(3):231-234, 1973.
- 5381 HAMARTOMA OF THE CERVICAL REGION. (Fr.) Manigand, G. (Bicetre Hosp., France), J. Paillas, D. Foulon, and M. Deparis. *Ann Med Interne (Paris)* 124(5):433-436, 1973.
- 5382 HERE ARE THE CURRENTLY MOST CONTROVERSIAL PROBLEMS CONCERNING MULTIPLE MYELOMA. (It.) Bufano, M. (Sch. Specialization Clin. Lab. Hematol., Univ. Rome, Italy). *Minerva Med* 64(35): 1871-1884, 1973.
- 5383 EXPERIMENTAL POLYMORPHIC OLIGODENDROGLIOMAS AND GLIOMAS IN THE RAT. HISTOCHEMICAL CONTRIBUTION. (It.) Giordana, M. T. (Clin. Nervous Mental Dis., Univ. Turin, Italy). *Acta Neurol (Napoli)* 28(3):235-241, 1973.
- 5384 ENDODERMAL SINUS TUMORS IN TERATOMAS OF THE THYMUS. (Rus.) Ageev, A. K. (S. M. Kirov Acad. Military Med., Leningrad, USSR). *Opr Onkol* 19(5):89-90, 1973.
- 5385 HEREDITARY PREDISPOSITION TO GASTRIC CANCER (ON FAMILIAL CANCERS). (Fr.) Dubarry, J.-J. (St. Andre Hosp., Bordeaux, France). *Bull Acad Natl Med (Paris)* 156(24/25):765-768, 1973.
- 5386 THYROID CANCER WITH AN AMYLOID STROMA: FIRST CASE IN FRENCH-SPEAKING AFRICA (CHAD). (Fr.) Sirol, J. (No affiliation) and H. Brottes. *Bull Soc Pathol Exot* 65(6):900-905, 1972.
- 5387 ACTIVITY OF SOME OXIDATION-REDUCTION ENZYMES IN THE EPITHELIUM OF PATIENTS WITH PATHOLOGICAL CHANGES IN THE UTERINE CERVIX. (Rus.) Ezhova, L. S. (All Union Res. Inst. Obstet. Gynecol., Moscow, USSR). *Akush Ginekol (Mosk)* (1):44-48, 1973.
- 5388 CONTENT OF GLYCOGEN AND NEUTRAL MUCOPOLYSACCHARIDES IN UTERINE MYOMAS. (Rus.) Aksenova, T. A. (Vladivostok Med. Inst., USSR). *Akush Ginekol (Mosk)* (1):16-18, 1973.
- 5389 EXCRETION OF GONADOTROPHIC HORMONES IN PATIENTS WITH UTERINE FIBROMAS. (Rus.) Benediktov, I. I. (Sverdlovsk Med. Inst., USSR) and N. A. Tron'. *Akush Ginekol (Mosk)* (1):13-15, 1973.
- 5390 STATUS OF SOME ENDOCRINE ORGANS IN PATIENTS WITH CERVICAL CANCER. (Rus.) Slepov, M. I. (V. I. Lenin Inst. Postgrad Med., Kazan, USSR). *Akush Ginekol (Mosk)* (3):43-46, 1973.
- 5391 SERUM L-LACTATE:NAD-OXIDOREDUCTASE ACTIVITY IN MALIGNANT TUMORS AND ITS RELATION TO THE SPREAD OF THE DISEASES. (Slov.) Jurga, L. (Med. Fac., P. J. Safarik U., Kosice, Czechoslovakia), L. Janocko, J. Andrasina and M. Klvana. *Bratisl Lek Listy* 59(4):428-433, 1973.
- 5392 HAIRY CELL LEUKEMIA: CLINICAL AND CYTOLOGICAL STUDY OF 55 CASES. (Fr.) Flandrinn, G. (St. Louis Hosp., Paris, France), M. T. Daniel, M. Fourcade and N. Chelloul. *Nouv Rev Franc Hematol* 13(5):609-640, 1973.
- 5393 MYXOMA IN THE RIGHT ATRIUM. (Cz.) Belobradek, Z. (Med. Fac., Charles U., Hradec Kralove, Czechoslovakia), I. Jurin, V. Pidrman and J. Prochazka. *Bratisl Lek Listy* 59(3): 349-356, 1973.
- 5394 "MALIGNANT" PAPILLOMATOSIS OF THE RESPIRATORY TRACT. (Fr.) Lamarche, J. (Maison-neuve-Rosemont Hosp., Montreal, Canada), G. Forget, D. Thibert and C. Auger. *Union Med Can* 102(8): 1687-1690, 1973.
- 5395 OSTEOSARCOMAS WHICH DEVELOPED IN RACHITIC BONES AFFECTED BY PAGET'S DISEASE: 4 CASES. (Fr.) Hauw, J. J. (Pitie-Salpetriere, U. Hosp. Ctr., Paris, France), D. Henin, G. Chomette and R. Escourolle. *Arch Anat Pathol (Paris)* 21(3): 241-249, 1973.
- 5396 BREAST TUMOR WITH MYOEPITHELIAL CELLS: ULTRASTRUCTURAL STUDY. (Fr.) Kermarec, J. (U. Hosp. Ctr., Nice, France), S. Plouvier, H. Duplay and R. Daniel. *Arch Anat Pathol (Paris)* 21(2):225-231, 1973.

5397 SOLITARY PLASMA CELL TUMORS OF THE BONE:
FOUR CASES. (Fr.) Calle, R. (Radium Inst.,
Paris, France), Y. Graic, A. Mazabraud and P.
Schlienger. *Bull Cancer (Paris)* 59(4):395-404, 1972.

5398 THE DIFFERENTIATION OF MURINE PLASMACYTOMA
MOPC 173: OBTAINING VARIANTS IN CELL CULTURE.
(Fr.) Guerin, C. (Inst. Molecular Biol., Fac. Sci.,
Paris, France), B. Prigent, M.-A. Moyne and A. Paraf.
Bull Cancer (Paris) 59(4):367-379, 1973.

5399 ONE CASE OF A SOLITARY PLASMA CELL TUMOR OF
THE BONE. FIRST CASE OBSERVED AT FORT LAMY
(CHAD) IN A NATIVE. (Fr.) Sirol, J. (No affiliation),
R. Laroche and D. Huot. *Bull Soc Pathol Exot* 65(6):
893-900, 1972.

5400 SUPRATENTORIAL HEMANGIOBLASTOMA OR "ANGIO-
BLASTIC" MENINGIOMA: ONE CASE. (Fr.)
Babin, P. (U. Hosp. Ctr., Poitiers, France), R.
Escourolle, J. P. Lefevre, J. J. Hauw, R. Gil and
P. de Giacomoni. *Arch Anat Pathol (Paris)* 21(3):267-
271, 1973.



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BIANCIFIORI, C. 4870*	BOYER, J. 4808	BURR, I.M. 5236*
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BISHUN, N.P. 4874*	BRAIN, M.C. 5201*	BUSSOLATI, G. 5251*
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- MILLER, R., JR.
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MORANO, E. 5209*	NAKAMURA, R.M. 5118*	ODASHIMA, S. 4886*
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MOULINIER, J. 4993	NATAF, R. 5168*	OKADA, K. 5192
MOULINIER, M.J. 5035*	NAVONE, R. 5121*	OKAMOTO, T. 4824
MOUNTAIN, I.M. 5098*	NAZERIAN, K. 5010	OLINICI, C.D. 5278*
MOUTQUIN, J.M. 5369*	NEDELEA, M. 5347*	OLIVIER, L. 5364*
MOVSESIAN, S.N. 4878*	NEGLEY, G.Z. 5338*	O'NEILL, C.H. 4966
MOVSESIAN, K.S. 5082*	NELSON-REES, W. 4954	O'NEILL, J. 5236*
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PATEL, S.B. 5193	POLOUVIER, S. 5396*	RAIN, B. 5107*
PATTEN, S.F. 4950	PODWORSKI, H. 5130*	RAKSHIT, M.M. 5220*
PATTON, G.W. 5318*	POLLARD, M. 5018	RALPH, P. 4865
PEARSON, G. 4940	PONTEFRAC, R.D. 4825	RAM, M.D. 5101*
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PEGNUM, S.M. 4860	PORTEOUS, D.D. 5086*	RAO, U.N.M. 5193
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PERONACE, M.L. 4898*	PRAGER, M.D. 5089*	REED, R.J. 5250*
PERRIN, J.-J. 5352*	PRAHL, B. 5226*	REGOECZI, E. 5201*
PERRON, L. 5370*	PRANG, L. 5223*	REIT, R. 5303*
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PESKIN, G.W. 5293*	PRECHTEL, K. 4838	REITZ, I. 4879*
PETERS, W.P. 4953	PREIN, J. 5281*	REMACLE-BONNET, M. 5362*
PETIT, J.-C. 5376*	PRICE, M.R. 5103*	RICHARDSON, J.R. 5274*
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PHILLIPS, M.J. 5328*	PRITCHARD, J.A.V. 5093*	RIESCO, A. 5368*
PICKEL, H. 5127*	PROCHAZKA, J. 5393*	RIGATTI, P. 5257*
PIDRMAN, V. 5393*	PROFISI-CENTA, G. 5175	RIGENBERG, L.K. 5243*
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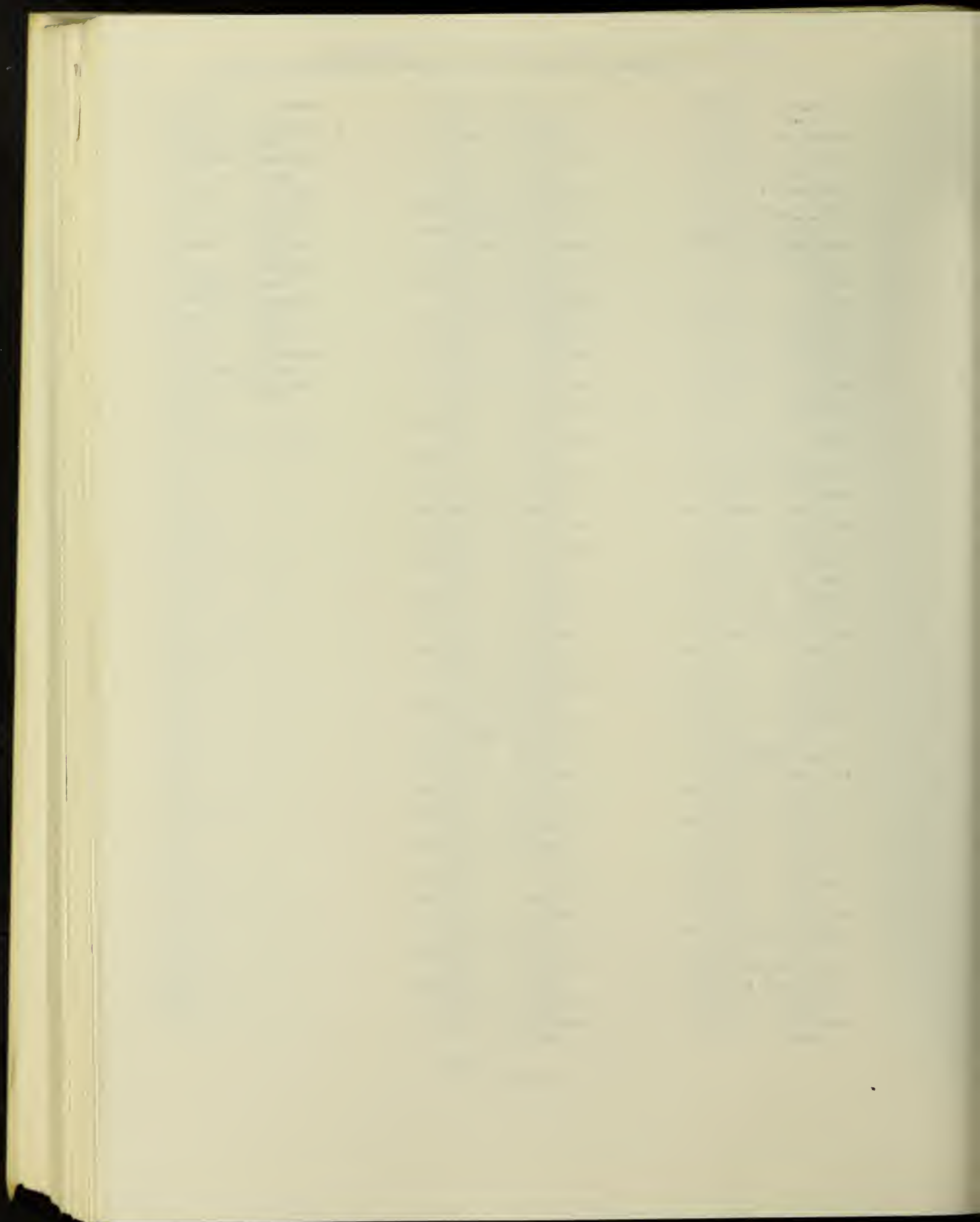
RIMM, A.A. 5070*	RUDIKOFF, S. 5078*	SCHAEFER, U.W. 4918*
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WAYSS, K. 5216*, 5218*	WIZNITZER, T. 5273*	ZAHN, R.K. 4977*
WEBER, H. 5292*	WOLF, R.H. 5267*	ZASUKHINA, G.D. 4991*
WEDDERBURN, N. 5005	WOLFF, G. 5226*	ZBAR, B. 5046*
WEISBURGER, E.K. 4837	WOLFF-TERROINE, M. 5314*	ZELENIN, A.V. 5227*
WEISBURGER, J.H. 4837	WOLFDORF, R.G. 4837	ZEYLEMAKER, W.P. 5048*
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WEISSMANN, C. 5200*, 5292*	WRBA, H. 4807	ZOTTER, S. 5000, 5108*
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WILLIAMS, D.C. 4812*, 4874*	YAMANISHI, Y. 5234*	
WILLIAMS, G.A. 4985*	YAMASHITA, T. 4969	
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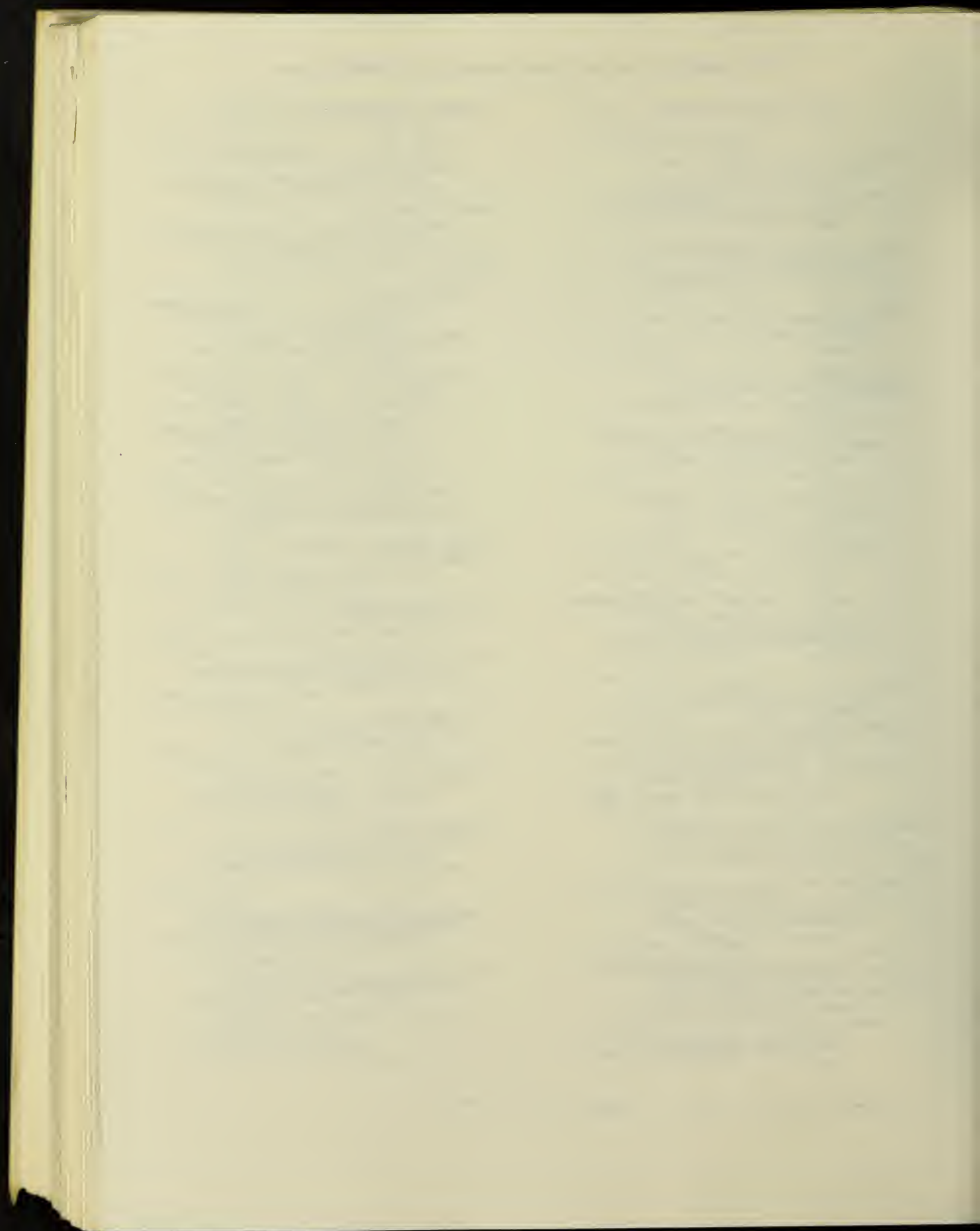
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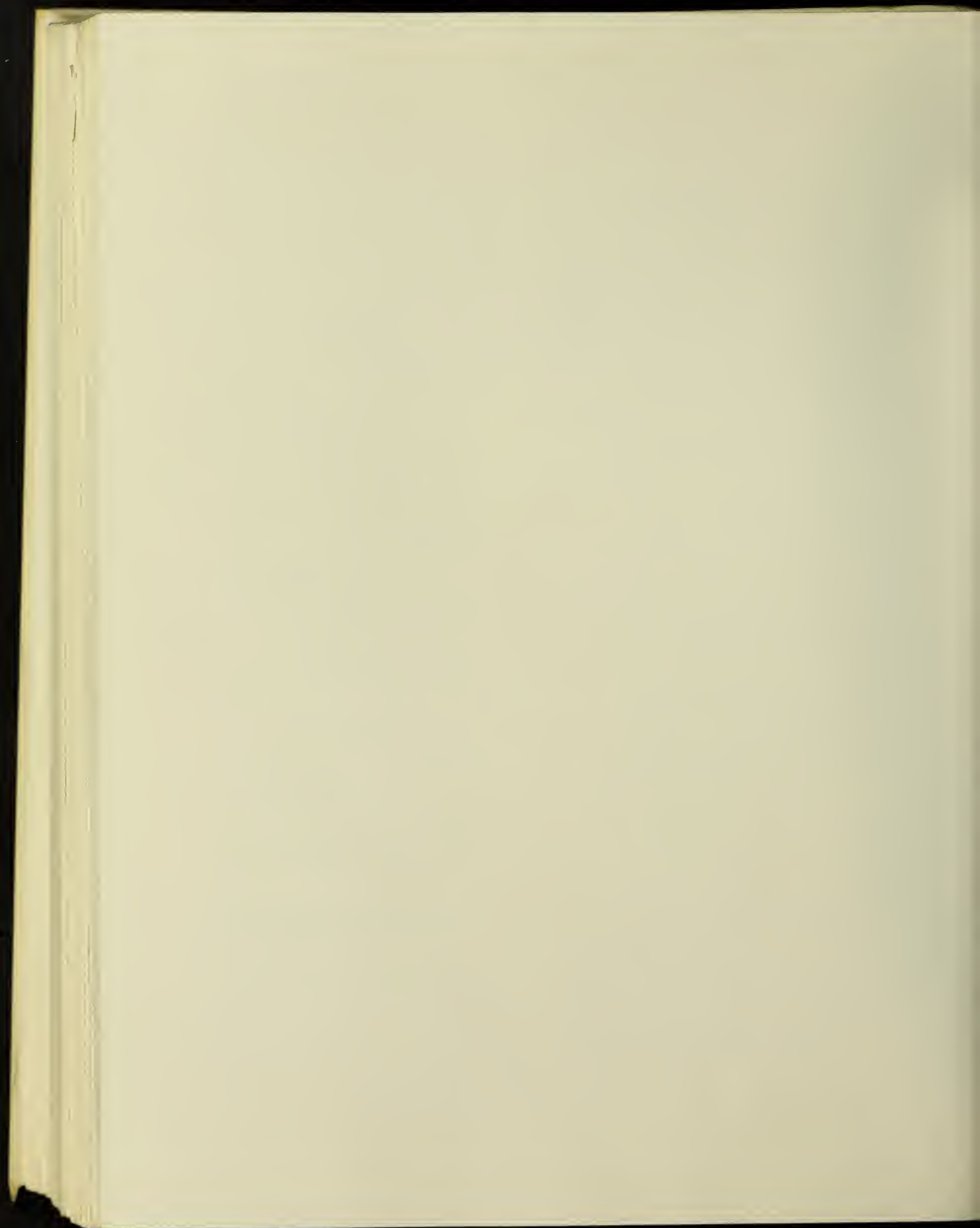
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